DEMOGRAPHIC, CLINICAL, INVESTIGATIONAL AND ETIOLOGICAL PROFILE OF PATIENTS WITH ACUTE SYMPTOMATIC SEIZURES

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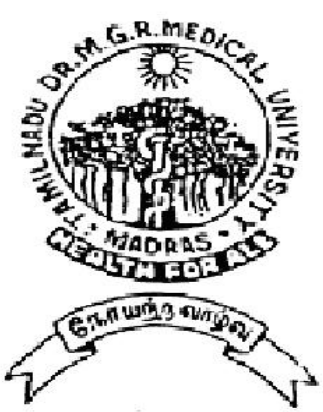

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CERTIFICATE

This is to certify that this dissertation titled "DEMOGRAPHIC, CLINICAL,INVESTIGATIONAL AND ETIOLOGICAL PROFILE OF ACUTE SYMPTOMATIC SEIZURES" submitted by DR.M.RADHAMANI to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of MD degree branch I General Medicine, is a bonafide research work carried out by her under our direct supervision and guidance.

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DECLARATION

I, Dr.M.RADHAMANI, solemnly declare that the dissertation titled "DEMOGRAPHIC , CLINICAL , INVESTIGATIONAL AND ETIOLOGICAL PROFILE OF ACUTE SYMPTOMATIC SEIZURES" has been prepared by me during the period of June 2008-June 2009 at Govt Rajaji Hospital. This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the regulations for the award of MD degree (branch I) General Medicine.

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CONTENTS

ABBREVIATIONS

GTCS	Generalised Tonic clonic Seizures
EPC	Epilepsia Partialis Continua
CT	Computed Tomography
MRI	Magnetic Resonance Imaging
MRV	Magnetic Resonance venography
SAH	Subarachnoid haemorrhage
ICH	Intra Cerebral Haemorrhage
TB	Tuberculosis
ANA	Anti Nuclear Anti body
dsDNA	Double standard Deoxy ribo Nucleic Acid
APLA	Anti Phospholipid Antibody
CSOM	Chronic Suppurative Otitis Media
NA	Not Applicable
CVT	Cerebral Venous Thrombosis
SSS	Superior Sagittal Sinus
LS	Lateral Sinus
SS	Sagittal Sinus

FND	Focal neurological deficit
HIV	Human immunodeficiency virus
NCC	Nuerocysticercosis
RHD	Rheumatic heart disease
CAD	Coronary artery disease
CKD	Chronic kidney disease
HTN	Hypertension
DM	Diabetes mellitus
LVH	Left ventricular hypertrophy
AVM	Arteriovenous malformations
LFT	Liver function test
ECG	Electrocardiogram
EEG	Electroencephalogram
CNS	Central nervous system
SCTEL	Single CT enhancing lesion
SSCCCTL	Small single cerebral calcific CT lesion

INTRODUCTION

Acute symptomatic seizures are those caused or provoked by an acute medical or neurological insult and it is as common as epilepsy in the medical wards. Acute symptomatic seizures shows a clearly identified causal association, generally tend not to recur , and usually long term antiepileptic treatment is not necessary . It may be single or repetitive. Type of seizure can be focal with or without generalisation or generalised. Most common is generalised tonic clonic seizure. Non convulsive seizure and status are common in patients admitted to intensive care units. Partial seizure is usually associated with structural abnormalities of brain but generalised seizure may result from cellular or structural abnormalities that have a wide spread distribution. In developing countries most common cause of acute symptomatic seizure is CNS infections. Seizures in the elderly may be caused by stroke, systemic metabolic conditions, subdural hematoma, central nervous system infection, degenerative disorders, or malignancy. The etiology of seizures is multifactorial in any given individual and is best thought of as an interaction between genetically determined seizure thresholds, underlying predisposing pathologies or metabolic derangements and acute precipitating factors.

About 5% of persons with head trauma, cerebrovascular disease and CNS infections have acute symptomatic seizures. The occurrence of acute symptomatic seizures is associated with an additional increase in risk for epilepsy. New-onset acute symptomatic seizures can be the presenting feature of

acute neurological diseases. The etiological spectrum of new- onset acute symptomatic seizures and outcome may be different in developing countries when compared to developed countries.

Approximately 60% of all epilepsies are idiopathic or cryptogenic. Almost any type of brain pathology can cause seizures. Cerebrovascular disease is the most commonly identified cause among elderly, while perinatal insults seem to be most common among children. Status epilepticus is one of the most common neurologic emergencies in children, adolescents, and young adults. Status epilepticus may be due to acute neurologic conditions such as meningitis, encephalitis, or stroke, complicated febrile seizures, intractable epilepsy, degenerative diseases, intoxication, or may be the first manifestation of epilepsy.

Very few studies are there, analysing the causes of acute symptomatic seizures, from south India . So it is useful to study the various conditions producing seizures in our patients and the use of investigations to find out the underlying problem. A detailed medical history, a thorough physical examination, especially of the nervous system, analysis of blood and other body fluids, electroencephalographic (EEG) recordings, magnetic resonance imaging (MRI) and/or computerized tomography (CT) scans we are able to find out the underlying cause of acute symptomatic seizures.Accurate diagnosis of the cause of acute symptomatic seizure is very important in the treatment and prognosis as the treatment of underlying condition will abolish the seizure .

REVIEW OF LITERATURE

A seizure is a paroxysmal event due to abnormal, excessive, hypersynchronous discharges from an aggregate of central nervous system neurons. Depending on the distribution of discharges, this abnormalCNS activity can have various manifestations, ranging from dramatic convulsive activity to experiential phenomena not readily discernible by an observer . Although a variety of factors influence the incidence and prevalence of seizures , ~5–10% of the population will have at least one seizure, with the highest incidence occurring in early childhood and late adulthood ([1]).

Epilepsy describes a condition in which a person has recurrent seizures due to a chronic , underlying process . This definition implies that a person with a single seizure , or recurrent seizures due to correctable or avoidable circumstances, does not necessarily have epilepsy. Epilepsy refers to a clinical phenomenon rather than a single disease entity , since there are many forms and causes of epilepsy . However, among the many causes of epilepsy there are various epilepsy syndromes in which the clinical and pathologic characteristics are distinctive and suggest a specific underlying etiology.

Seizures have been classified in several ways: according to their supposed etiology, i.e., idiopathic (primary) or symptomatic (secondary);their site of origin; their clinical form (generalized or focal); their frequency

(isolated, cyclic, or repetitive, or the closely spaced sequence of status epilepticus); or their electrophysiologic correlates([2]). The classification of seizure was first proposed by Gastaut in 1970 and was then refined repeatedly by the Commission on Classification and Terminology of the International League Against Epilepsy (1981). This classification, based mainly on the clinical form of the seizure and its electroencephalographic (EEG) features, has been adopted worldwide and is generally referred to as the International Classification([2]). A simpler classification system, another ILAE classification that was developed in 1993 for conducting epidemiological survey on epilepsy. This was named the Epidemiological Classification (EC) and was proposed for only research purposes to overcome technical problems in field studies.

Acute symptomatic seizure was defined as seizure caused or provoked by an acute medical or neurological insult. Acute symptomatic seizures were further grouped into two broad categories: 1) acute symptomatic seizure caused by acute neurological insult; and 2) acute symptomatic seizure caused by acute metabolic disorder([3]).

Classification of seizures[(56)]

1.GENERALIZED SEIZURES

Tonic clonic (in any combination)

Absence Typical

Atypical

Absence with special features

Myoclonic absence

Eyelid myoclonia

Myoclonic Myoclonic

Myoclonic atonic

Myoclonic tonic

Clonic

Tonic

Atonic

2. FOCAL SEIZURES

* Without impairment of consciousness/responsiveness

+ With observable motor or autonomic components (simple partial seizure)

+ Involving subjective sensory or psychic phenomena only (aura)

* With impairment of consciousness/responsiveness (complex partial seizure)

* Evolving to a bilateral, convulsive seizure (replaces the term secondarily generalized seizure)

3.MAY BE FOCAL, GENERALIZED, OR UNCLEAR Epileptic spasms

Outline of the Epidemiological Classification Commission on Epidemiology and Prognosis, International League Against Epilepsy[56].

2.1. Generalized seizures – when clinical symptomatology provides no indication of an anatomic localization and no clinical evidence of focal onset.

2.2 Partial seizure – when there is evidence of a clinical partial onset (by aura or focal symptoms).

2.3/2.4 Undetermined seizures – it is impossible to classify seizures owing to lack of adequate information or variable/mixed partial and generalized seizures.

Risk factors or etiology

3.1 Symptomatic seizures or epilepsies – consequence of a known cerebral dysfunction

3.1.1 Acute symptomatic – seizures are in close temporal association (within 7 days) with an acute systemic, metabolic or toxic insult and with acute CNS insult (infection, stroke, cranial trauma, intracerebral hemorrhage, acute alcohol intoxication or withdrawal).

3.1.2 Remote Symptomatic – seizures in relation to a well demonstrated antecedent condition called as remote symptomatic seizures or epilepsy (more than 7 days of cerebral insult).

3.2 Unknown etiology – no clear antecedent etiology can be detected .

Basically, this classification divides seizures into two types—partial, in which a focal or localized onset can be discerned, and generalized, in which the seizures appear to begin bilaterally. It is also useful clinically and etiologically to separate epilepsies that originate as truly generalized electrical discharges in the brain from those which spread secondarily from a focus to become generalized. The primary generalized epilepsies are a group of somewhat diverse, age-dependent phenotypes that are characterized by generalized 2.5- to 4-Hz bifrontally predominant spikes or polyspike-and-slow-wave discharges that arise without underlying structural abnormalities(2).

Seizures that begin locally and evolve into generalized tonic-clonic seizures, termed secondary generalized seizures, generally have no such genetic component and are usually the result of underlying brain disease, either acquired or due to congenital malformations or metabolic defects.Partial seizures vary with the locale of the lesion and are conventionally divided into two groups, simple and complex, depending on whether consciousness is retained or impaired. Simple partial seizures most often arise from foci in the sensorimotor cortex. Complex partial seizures most often have their focus in the temporal lobe on one side or the other, but a frontal localization is also well known . If the seizure lasts more than 30 minutes or there is sequential seizure without recovery of conciousness it is called status epilepticus . Epilepsia Partialis Continua is a special type of focal motor epilepsy characterized by persistent rhythmic clonic movements of one muscle group—usually of the

face, arm, or leg—which are repeated at fairly regular intervals every few seconds and continue for hours, days,weeks, or months without spreading to other parts of the body. It is a highly restricted and very persistent focal motor status epilepticus(2).

The first solitary seizure or brief outburst of seizures may occur during the course of many medical illnesses. It indicates that the cerebral cortex has been affected by disease, either primarily or secondarily. Convulsive seizures by their nature, if repeated every few minutes, as in status epilepticus, may threaten life. Equally important, a seizure or a series of seizures may be the manifestation of an ongoing neurologic disease that demands the employment of special diagnostic and therapeutic measures .

The age of the patient greatly affects the incidence of certain seizure types; e.g., absence and myoclonic seizures are relatively more common in children and adolescents. Furthermore, the underlying causation varies greatly by age([2]). Adolescence (10–18years) common causes are idiopathic epilepsy, including genetically transmitted types, juvenile myoclonic epilepsy, trauma and drugs and in early adulthood(18–25 years) causes are idiopathic epilepsy,trauma, neoplasm,withdrawal from alcohol or other sedative drugs. Middle age (35–60years) trauma, neoplasm, vascular disease,alcohol or other drug withdrawal are main etiology. Late life (over 60years) vascular disease (usually post -infarction),tumor, abscess, degenerative disease and trauma takes main causes.

History from a reliable attender is very important in the diagnosis of seizure.Type of seizure , risk factors , predisposing events,and past illness should be obtained from the history to determine its pattern and other characteristics; and undertake a search for its cause. In the diagnosis of epilepsy history is the key and the physical examination is important to identify any underlying etiology .

The general physical examination includes a search for signs of infection or systemic illness. Careful examination of the skin may reveal signs of neurocutaneous disorders, such as tuberous sclerosis or neurofibromatosis, or chronic liver or renal disease. A finding of organomegaly may indicate a metabolic or storage disease, and limb asymmetry may provide a clue to brain injury early in development. Signs of head trauma and use of alcohol or illicit drugs should be sought. Auscultation of the heart and carotid arteries may identify an abnormality that predisposes to cerebrovascular disease[1,2].

All patients require a complete neurologic examination, with particular emphasis on eliciting signs of cerebral hemispheric disease([1,2]).Careful assessment of mental status (including memory, language function, and abstract thinking) may suggest lesions in the anterior frontal, parietal, or temporal lobes.

Testing of visual fields will help screen for lesions in the optic pathways and occipital lobes. Screening tests of motor function such as pronator drift, deep tendon reflexes, gait, and coordination may suggest lesions in motor

(frontal) cortex, and cortical sensory testing (e.g., double simultaneous stimulation) may detect lesions in the parietal cortex.

LABORATORY STUDIES

Routine blood studies are indicated to identify the more common metabolic causes of seizures, such as abnormalities in electrolytes, glucose, calcium, or magnesium, and hepatic or renal disease. A screen for toxins in blood and urine should also be obtained from all patients in appropriate risk groups, especially when no clear precipitating factor has been identified. A lumbar puncture is indicated if there is any suspicion of meningitis or encephalitis, and it is mandatory in all patients infected with HIV, even in the absence of symptoms or signs suggesting infection[(1).]

ELECTROPHYSIOLOGICAL STUDY

All patients who have a possible seizure disorder should be evaluated with an EEG as soon as possible.In the evaluation of a patient with suspected epilepsy, the presence of electrographic seizure activity during the clinically evident event clearly establishes the diagnosis [(1,2)]. The absence of electrographic seizure activity does not exclude a seizure disorder, however, because simple or complex seizures may originate from a region of the cortex that is not within range of the scalp electrodes. The EEG is always abnormal during generalized tonic-clonic seizures. Since seizures are typically infrequent and unpredictable, it is often not possible to obtain the EEG during a clinical event. Continuous monitoring for prolonged periods in video-EEG telemetry

units for hospitalized patients or the use of portable equipment to record the EEG continuously on cassettes for 24 h in ambulatory patients has made it easier to capture the electrophysiologic accompaniments of clinical events. In particular, video-EEG telemetry is now a routine approach for the accurate diagnosis of epilepsy in patients with poorly characterized events or seizures that are difficult to control[8].

Magnetoencephalography (MEG) provides another way of looking noninvasively at cortical activity, it measures the small magnetic fields that are generated by this activity. Epileptiform activity seen on the MEG can be analyzed, and its source in the brain can be estimated using a variety of mathematical techniques. These source estimates can then be plotted on an anatomic image of the brain, such as an MRI to generate a magnetic source image (MSI). MSI can be useful to localize potential seizure foci[9].

The EEG may also be helpful in the interictal period,shows epileptiform activity . The presence of epileptiform activity is not specific for epilepsy, but it has a much greater prevalence in patients with epilepsy than in normal individuals. However, even in an individual who is known to have epilepsy, the initial routine interictal EEG may be normal up to 60% of the time. EEG is used for classifying seizure disorders and aiding in the selection of anticonvulsant medications. Focal interictal epileptiform discharges would support the diagnosis of a partial seizure disorder such as temporal lobe epilepsy or frontal lobe seizures, depending on the location of the discharges.Also used

to assess the prognosis of seizure disorders; in general, a normal EEG implies a better prognosis, whereas an abnormal background or profuse epileptiform activity suggests a poor outlook[16]. The EEG has not proved to be useful in predicting which patients with predisposing conditions , such as head injury or brain tumor, will go on to develop epilepsy, because in such circumstances epileptiform activity is commonly encountered regardless of whether seizures occur.

BRAIN IMAGING

Almost all patients with new-onset seizures should have a brain imaging study to determine whether there is an underlying structural abnormality that is responsible[10]. MRI has been shown to be superior to CT for the detection of cerebral lesions associated with epilepsy. In some cases MRI will identify lesions such as tumors, vascular malformations, or other pathologies that need immediate therapy. The use of newer MRI methods, such as fluid-attenuated inversion recovery(FLAIR), has increased the sensitivity for detection of abnormalities of cortical architecture, including hippocampal atrophy associated with mesial temporal sclerosis, as well as abnormalities of cortical neuronal migration. In such cases the findings may not lead to immediate therapy, but they do provide an explanation for the patient's seizures and point to the need for chronic anticonvulsant therapy or possible surgical resection[11,12].

In the patient with a suspected CNS infection or mass lesion, CT scanning should be performed emergently when MRI is not immediately available. Functional imaging procedures such as positron emission tomography (PET) and single photon emission computed tomography (SPECT) are also used to evaluate certain patients with medically refractory seizures. Volumetric MR Imaging gives quantitative evaluation of hippocampal volume has been found to marginally increase the sensitivity over visual analysis in detection of hippocampal sclerosis. T2 Relaxometry is used to quantify the T2 signal in the hippocampus , in mesial temporal sclerosis the relaxation time has proven to be lengthened by 10 milliseconds. MR Spectroscopy has been widely used in providing insight into the metabolic alterations in epilepsy [(12)].

EPIDEMIOLOGY

Epidemiology of acute symptomatic seizure varies in different age groups and in different countries . Developing countries major cause is CNS infections .A study done at a university hospital in South India[(4)]showed that seizure occurred in close temporal association with an acute central nervous system (CNS) insult in 53% of patients. Cerebrovascular diseases were the risk factors in 48% of patients with remote symptomatic epilepsy. Infections of CNS including single CT enhancing lesion (SCTEL) accounted for 77% of patients with acute symptomatic epilepsy. Neurocysticercosis, SCTEL and small single cerebral calcific CT lesion (SSCCCTL) together accounted for 40% of etiological factors and neurotuberculosis for 10%. Infections of the central

nervous system and SCTEL together were the putative risk factors in 52% of patients aged ≤40 years. The type of seizure was either simple partial or complex partial with or without secondary generalization in 76% of patients.

Another study from south India [6]the etiological risk factors were central nervous system infections in 32% patients, metabolic disorders in32%, cerebrovascular diseases (ischemic, venous and hemorrhagic) in 21% and others in 15%. The distribution of the pathology in patients with CNS infections was meningoencephalitis in 43% and parenchymal granuloma in 57% of patients, in that degenerative phase solitary cystic granuloma in 75% and tuberculoma in 25% of patients.

Study by Sander et al[13] 20% had febrile seizures,52% definite epileptic seizures, and 21% possible epilepsy. In the definite epilepsy group the proportions of males and females were similar, 25% (21-28%) were younger than 15 years and 24% (21-28%) were 60 years or older. The definite seizures were classified as cryptogenic in 62% (58-66%), remote symptomatic in 21% (18-25%), and acute symptomatic in 15% (12-18%). The aetiology of epilepsy was vascular disease in 15% (12-18%) and tumor in 6% (4-8%). Among older subjects the proportion with an identifiable cause was much higher: 49% (41-58%) were due to vascular disease and 11% (6-16%) to tumor[13].

Developed countries most common cause is cerebrovascular disease. Cerebrovascular disease may account for ~50% of new cases of epilepsy in patients older than 65. Acute seizures i.e., occurring at the time of the stroke are

seen more often with embolic rather than hemorrhagic or thrombotic stroke. Chronic seizures typically appear months to years after the initial event and are associated with all forms of stroke.

The reported incidence of post-stroke seizures varies widely between epidemiological studies, ranging from 2% to 33% for early seizures and 3% to 67% for late seizures[5] . According to Joseph et al cerebrovascular disease is the most commonly identified cause of acquired epilepsy. Post-stroke seizures account for 11% of all epilepsy, 22% of all cases of status epilepticus, and 55% of newly diagnosed seizures amongst older patients. According to Maurizio et al the risk of first seizure was increased in cortical involvement, multiple CT-scan lesions, supratentorial lesions, prior lesions on CT-scan, family history of seizures, use of epileptogenic drugs, large lesions, hemorrhagic lesions, and cortical atrophy.

Patients with CVT present with varying combinations of headache, seizures, aphasia, behavioral abnormalities, altered sensorium and focal deficits. Seizures may be focal, multi focal or generalized. The presentation is acute in obstetric and infectious CVT while a slowly progressive disease is more common in inflammatory and idiopathic cases. Thrombosis involves the dural sinuses as well as cortical veins producing cerebral infarction and neurological deficit .

Seizures that begin in patients of adolescence and early adulthood may be associated with head trauma, CNS infections including parasitic infections such

as cysticercosis, brain tumors,congenital CNS abnormalities, illicit drug use, or alcohol withdrawal.

Head trauma is a common cause of epilepsy in adolescents and adults. A patient with a penetrating head wound, depressed skull fracture, intracranial hemorrhage, or prolonged post-traumatic coma or amnesia has a 40-50% risk of developing epilepsy, while a patient with a closed head injury and cerebral contusion has a 5–25% risk. Recurrent seizures usually develop within 1 year after head trauma, although intervals of 10 years are well known. In controlled studies, mild head injury , defined as a concussion with amnesia or loss of consciousness of <30 min, was found to be associated with only a slightly increased likelihood of epilepsy.

Infections are important cause of seizures in developing countries, the frequency of which may differ widely in different locations. Viral, bacterial, fungal and parasitic infections can result in seizures.

Viral infection of the brain may cause either aseptic meningitis or encephalitis. The virus causing aseptic meningitis includes entero-virus(more common in developing countries because of faecal-oral transmission), mumps, arena viruses, herpes simplex type-2, varicella zoster and HIV[(2)].

Herpes simplex encephalitis has no pathognomonic clinical presentation but presents as focal encephalitis with malaise; focal seizures that may become generalized. Herpes simplex encephalitis produces dramatic ectroencephalographic (EEG) focal ,temporal or lateralized polymorphic delta

activity as the earliest Changes. CT and MRI reveal medial temporal involvement.[14]

In Japanese encephalitis convulsions may occur as part of severe encephalitis and, the mortality rates are high (20% to 40%). MRI shows thalamic and basal ganglia involvement. About 10% of the patients may show a bi-phasic illness pattern in which the seizures are more common during first or the acute phase of the illness[5,14].

Seizures are not uncommon in patients with human immunodeficiency virus (HIV) infection, and with the upsurge in HIV infection this may be an important cause for acute symptomatic seizures. Seizures may rarely be the presenting manifestation of HIV infection.Opportunistic infections such as toxoplasmosis, tuberculosis, progressive multifocal leucoencephalopathy (PML), cryptococcal meningitis and polymicrobial infections, metabolic and electrolyte disturbances, and drugs are common causes of new-onset seizures in HIV. In the absence of any cause, primary HIV infection maybe considered responsible for seizures . The treatment of HIV-infected individuals with seizures comprises of the administration of AEDs, specific treatment of the underlying conditions, and antiretroviral drugs.

Neurocysticercosis is a disease caused by the infection with the larval stage of the intestinal cystode Taenia solium that occurs when human or porcine become intermediate hosts. The parasite has marked tendency to infect muscle and the central nervous system where it produces a pleomorphic clinical

disorder neurocysticercosis . In many developing countries neurocysticercosis is the most common parasitic disease of the central nervous system and accounts for 10% of all acute neurological diseases. There are wide variations of clinical manifestations of neurocysticercosis. These are consequence of inflammation around a cyst, space occupation and impedance to the flow of cerebrospinal fluid. Less commonly, there is meningeal or vascular inflammation. Epilepsy is the most common manifestation of neurocysticercosis, occurring in two-third of affected patients.[3,4] Acute symptomatic seizures occur during the focal encephalitic illness caused by degenerating parasite but chronic epileptogenic focus that causes late epilepsy develops due to healing by peri-lesional gliosis and chronic calcified lesion . Seizures in neurocysticercosis are generalized convulsive or simple partial with focal motor activity.

Medina et al. in their series of 50 patients with epilepsy due to neurocysticercosis [13] found that 72% had partial seizures. Del Brutto et al. studied clinical characteristics of 203 patients with epilepsy and neurocysticercosis and found generalized convulsive seizures in 38% and complex partial seizures in 2%.

Neuro-imaging is essential to the diagnosis of neurocysticercosis. Brain MRI is superior for showing intraventricular or subarachanoid cyst,and for showing inflammation around a cyst whereas CT is better for showing the calcification of inactive lesions. However, recent MRI studies with gradient echo and reversed gradient echo phase image have shown scolex visible within the

calcified lesion as visible on CT scan; these entrapped antigens have been shown to be responsible for intermittent immuno-allergic response, peri-lesional edema and seizure recurrence. There may be single or multiple cysts in different pathological stage[15].

Carpio has proposed a classification system that corresponds to the viability of the parasite: active, transitional and inactive. Both CT and MRI can show the presence of the eccentric mural nodule (the invaginated scolex), an appearance when multiple is pathognomonic of neurocysticercosis(starry night appearance). Magnetization transfer spin echo sequence of MRI and calculation of magnetization transfer ratios used to differentiate neurocysticercosis from tuberculoma[16].

Malaria is the most common fatal parasitic disease . Approximately 2% of all patients with malaria have cerebral involvement and nearly 80% of patients who ultimately die have cerebral involvement. Cerebral malaria is fatal in 20% to50% of affected patients. In a recent report from Nigeria where malaria is endemic, cerebral malaria is responsible for one third of seizures with fever in childhood[16].

In immunocompromized patients, cerebral toxoplasmosis produces nonspecific signs and symptoms of intracranial mass lesion and seizures. In the developing world toxoplasmosis may occur without HIV infection as well and linear beaded appearance on MRI may be diagnostic . Focal neurological deficits occurred in (69%) and seizure in (29%).

Brain tuberculomas make up 5 to 8 per cent of intracranial masses in person in developing countries[17]. Before effective chemotherapy was available for tuberculosis, tuberculoma made up 20 per cent of intracranial lesions in one large series[19]. The incidence of neurotubeculosis in the United States is less than 0.5 per cent[18]. Tuberculomas are granulomatous mass lesions composed of a central zone of caseation surrounded by a collagenous tissue capsule arising in the brain parenchyma or the spinal cord. The commonest presenting symptoms [20] were headache (100%), partial or generalized convulsions (68.7%) and hemiparesis with or without hemisensory symptoms (56.2%).The role of neuro-imaging by CT or MRI scan in the diagnosis of tuberculoma is well-established . MRI is superior to CT in visualizing the morphological details of tuberculoma, and particularly the tiny brain stem lesions. Improvements in diagnostic efficacy have been made possible by utilizing MR diffusion weighted imaging, spectroscopy and minimally invasive CT-guided biopsy. The few studies available from developing countries on the frequency of radiological clearance have shown complete resolution of the intracranial lesions in 80–100% of patients on short-course (6–12 months) chemotherapy. A recent study from India, including only histopathologically verified cases, revealed a lower rate of 54% complete resolution by 24 months of treatment [20] . According to Jayasree et al [6] 32% of cases of seizures were due to CNS infections , of this 4.5%contributed by tuberculoma .

Seizure is a common complication of cerebral abscess, frequently occurring at presentation. Early seizures predispose to late seizures and in these patients long-term anticonvulsant treatment should be considered. There is no relationship between the site of the abscess, organism cultured, surgical treatment, presumed aetiology, age or sex of the patient and seizure occurrence.

Metabolic disturbances such as electrolyte imbalance, hypo- or hyperglycemia, renal failure, and hepatic failure, hypernatremic hyperosmolar state, thyrotoxic storm, porphyria , hypomagnesemia,and hypocalcemia may cause seizures at any age. Rapidly evolving electrolyte abnormalities are more likely to cause seizures than those occurring gradually. Similarly, endocrine disorders, hematologic disorders, vasculitides, and many other systemic diseases may cause seizures over a broad age range. A wide variety of medications and abused substances are known to precipitate seizures [(1)].

Diabetic nonketotic hyperosmolar state manifests with diverse neurological manifestations that include clouded sensorium, partial motor seizures, transient hemiplegia, chorea, hemiballismus and hemichorea – hemiballismus[(24)].

Patients with renal failure may manifest a variety of neurologic disorders. Patients with chronic renal failure who have not yet received dialysis therapy may develop a symptom complex progressing from mild sensorial clouding to delirium and coma, with tremor, asterixis, multifocal myoclonus, and seizures. After the institution of adequate maintenance dialysis therapy, patients may continue to be afflicted with more subtle nervous dysfunction, including

impaired mentation, generalized weakness, and peripheral neuropathy. These central nervous system disorders are referred to as uremic encephalopathy. The dialysis treatment of end-stage renal disease has itself been associated with the emergence of two distinct, new disorders of the central nervous system; dialysis dysequilibrium and dialysis dementia. The dialysis disequilibrium syndrome consists of headache, nausea, muscle cramps, obtundation, and seizures, and is a consequence of the initiation of dialysis therapy in some patients . A study[26] of nine patients, aged 23 to 67 years, showed a remarkable sequence of EEG findings in progressive uremic encephalopathy. The initial characteristics suggested a disorder of subcortical gray matter, followed by involvement of cortical gray matter and finally white matter. Seizures indicated a grave prognosis[26] .When seizures occur in the context of renal insufficiency, it is necessary to rule out a number of complications other than uremia :electrolyte imbalance(water intoxication,hypocalcemia,hyponatremia,hypomagnesemia) , aluminum encephalopathy ,drug intoxication,hypertensive encephalopathy , intracranial hemorrhage ,subdural hematoma and Wernicke's encephalopathy.

Aggravation of seizures due to hyponatremia was investigated in five patients with epilepsy and polydipsia–hyponatremia. They experienced marked increases in the frequency of their complex partial seizures with a decrease in the serum sodium level to 118–127 mEq/L . In all cases , the serum sodium level returned to normal through restriction of fluids, and the clinical seizures improved.

Seizure can occur in organophosphorus poisoning . incidence is 22-25% in children and 2-3% in adult . Electrographic seizures are a feature of organophosphate anticholinesterase intoxication[33]. Clinical studies of pesticide poisonings suggest that seizures are more common in children than in adults. Since flaccid paralysis, a characteristic sign of organophosphate poisoning, can mask convulsions, the most reliable indicator of seizures is the electroencephalogram. Seizures can rapidly progress to status epilepticus, contributing to mortality and, in survivors, to neuronal damage and neurological impairment. Anticonvulsant drugs can significantly reduce the lethal and toxic effects of these compounds. A benzodiazepine, usually diazepam, is the treatment currently indicated for control of seizures. Neuropathology caused seizures is most likely associated with glutamatergic excitotoxicity. Future prospects for improved treatments include new benzodiazepines, glutamate receptor antagonists, antimuscarinics with additional antiglutamatergic activity and adenosine receptor antagonists.The illegal mixing of organophosphates and pyrethroids in marketed agriculture insecticides in developing countries cause combination of miosis, bradycardia, tachypnea, and unconsciousness and seizures. The occurrence of pupillary dilation after a small-dose infusion of atropine (0.08 to 0.2 mg/kg in 1–3 h) and seizures raise the possibility of pyrethroid poisoning.

Alcohol Withdrawal siezure is seen when an individual reduces or stops alcohol consumption after prolonged periods of excessive alcohol intake.

Approximately 23-33% of patients with significant alcohol withdrawal have alcohol withdrawal seizures ("rum fits").Seizures are usually brief, generalized, tonic-clonic in nature, and without an aura. They occur in a cluster of 1-3 seizures with a short postictal period. Partial seizures are not uncommon.The incidence peaks 24 hours after the most recent alcohol ingestion.Most seizures typically terminate spontaneously or are easily controlled with benzodiazepines. Status epilepticus may occur in 3% of alcohol withdrawal seizures and should prompt an investigation for other causes, as people with alcoholism are prone to head injuries, chronic idiopathic epilepsy, and meningitis.

Seizures affect 50% of patients with primary and metastatic brain tumors[34]. Partial seizures have the highest incidence, followed by secondarily generalized, depending on histologic subtype, location, and tumor extent. The underlying pathophysiologic mechanisms of tumor-associated seizures are poorly understood and include theories of altered peritumoral amino acids, regional metabolism, pH, neuronal or glial enzyme and protein expression, as well as immunologic activity. An involvement of changed distribution and function of N-methyl-d-aspartate subclass of glutamate receptors also has been suggested. The often unpredictable responses to seizures after surgical tumor removal add substantial evidence that multiple factors are involved. Studies are needed to elucidate more clearly the pathophysiologic mechanisms of tumor-related seizures and to identify and develop the optimal AEDs. Oligodendroglial brain tumours 75% presented with symptoms related to seizures[35].

TREATMENT

The treatment of seizures of all types can be divided into - the use of antiepileptic drugs, the surgical excision of epileptic foci and other surgical measures, the removal of causative and precipitating factors, and the regulation of physical and mental activity .Choices of antiepileptic drugs depends on type of seizure [2] and patient characteristics .Preferably start a conventional or first line AED like phenytoin ,phenobarbitone,carbamazepine,oxcarbamazepine ,or valproate .Newer AED's are lamotrigine,leviteracetam, topiramate,zonisamide, gabapentin, tiagabine and felbamate.

SEIZURE TYPE	INITIAL CHOICE	SECOND LINE
Tonic-clonic	Valproate,Phenytoin carbamazepine	Lamotrigine, Oxcarbazepine
Myoclonic	Valproate	Lamotrigine
Partial	Carbamazepine,phenytoin	ValporateLamotrigine, Oxcarbazine
Absence	Valproate	Ethosuximide, Lamotrigine
Unclassifiable	Valproate	Lamotrigine.

Treatment of the underlying condition along with AED is very important in patients with acute symptomatic seizures .Seizures due to metabolic and withdrawal states, treatment with anticonvulsants is usually not necessary as long as the underlying disturbance is rectified. Antiepileptic drug therapy should

be started in any patient with recurrent seizures of unknown etiology or a known cause that cannot be reversed. Initiating therapy in a patient with a single seizure is controversial . Patients with structural brain lesions like tuberculoma, NCC or single enhancing lesion brain tumor, vascular malformation, or brain abscess need AED until resolution of lesion and maintain on an antiepileptic medication for at least 1 year, and an attempt is made to withdraw medications only if the patient has been completely seizure-free[(1)]. If seizures are refractory to medication, the patient may benefit from surgical removal of the epileptic brain region .Single seizure after stroke may not be treated and if there is high risk for reccurence AED can be given . For patients with CVT use of AED is recommended for one year . Gabapentin , and lamotrigine are the first line drugs for post stroke seizure .Patients with brain tumors AED should be given before and after surgery . Patients with uremic seizures safer AED ‘s are lamotrigine,valproate and phenytoin .

Patients with tuberculoma or TB meningitis should be treated with ATT. 6month course is acceptable but should be treated 9-12 months in patients who have an inadequate resolution or positive culture during treatment . Steroids should be given to prevent complications . For brain abscess high dose parentral antibiotics and surgical drainage are advised . Neurocysticercosis is treated with albendazole 15mg/kg /d in 2 doses for 8 days or praziquantel 50mg/kg/d for 15 days . For patients with CVT anticoagulants initially intravenous followed by oral and antibiotics if it is septic thromboplebitis are

employed . Patients with tumor have to be treated surgically followed by radiotherapy or chemotherapy .

Patients with status epilepticus should be treated promptly as the condition is associated with high mortality and cardiorespiratory dysfunction, hyperthermia, and metabolic derangements can develop as a consequence of prolonged seizures, and these can lead to irreversible neuronal injury . Quick assessment of cardiorespiratory function and airway and insert large-bore intravenous line and draw blood for glucose, blood urea nitrogen, electrolytes, and a metabolic and drug screen. A normal saline infusion is begun and a bolus of glucose is given (with thiamine if malnutrition and alcoholism are factors). Diazepam is given intravenously at a rate of about 2 mg/min until the seizures stop or a total of 20 mg has been given. Or lorazepam, 0.1 mg/kg given by intravenous push at a rate not to exceed 2 mg/min . A loading dose 20 mg/kg of phenytoin is administered by iv at a rate of less than 50 mg/min or fosphenytoin at 150 mg/min.If the seizure is not controlled repeat phenytoin 7-10mg/kg.Consider sodium valproate 25mg/kg or phenobarbitone 20mg/kg infusion if the seizure still continues. Admit in ICU and give iv anaesthesia with midazolam or propofol as next step .Once the seizure is controlled continue maintanance AED .

AIMS AND OBJECTIVES

The aims of the study were as follows

1. To analyze the etiological factors in patients >12 years of age presenting with acute symptomatic seizures .

2. To study the incidence of potentially curable causes of seizures .

3. To study the pattern of seizures and associated features.

4. To study the usefulness of various investigations in the diagnosis of acute symptomatic seizures .

MATERIALS AND METHODS

THE STUDY GROUP

The study was conducted on patients admitted in medical wards and IMCU of Government Rajaji Hospital, Madurai. Approval from the hospital ethical committee was obtained.

STUDY DESIGN

The study was a cross sectional study conducted for a period of one year between June 2008- June 2009.

Inclusion criteria

1.Patients admitted with first episode of seizure .

2.Age more than 12 years .

3.Patients admitted for other medical conditions who develop seizure during hospital stay .

Exclusion criteria

1.Patients with previous history of seizure

2.Idiopathic seizures

METHODS

Consecutive patients with new-onset acute symptomatic seizure as the first presenting event with acute illness admitted to medical wards and IMCU of Government Rajaji Hospital, Madurai were studied . A total number of 154 patients were studied out of which 100 were included in the study as per inclusion and exclusion criteria . All the details were noted in a specially

prepared proforma a copy of which is annexed . All patients were from low socioecnomical status.A detailed history was elicited from the relatives about the type of seizure duration ,associated symptoms like fever ,headache, vomiting ,weakness or loss of consciousness . Past history of medical illness or neurological illness was elicited . Detailed examination especially neurological, was done to find out any etiological factors ,focal neurological deficits or complications . Fundus examination was done to look for papilledema, or retinopathy .Blood pressure was checked and categorised to different stages according to JNC 7.

Baseline investigations done to find out metabolic problems ,renal function, liver function and electrolyte imbalances . ECG was done for all patients to find out any cardoivascular abnormality .CSF examination done for indicated patients .Neuroimaging was done for all patients with seizures as an emergency mainly CT brain .MRI brain was done only if the CT is inconclusive or diagnosis is doubtful or if there was need for imaging of sinuses and venous system . CT or MR angiogram was done for patients with CVT , AVM or other vascular abnormalities . Special MRI sequences like FLAIR ,DWI, MR spectroscopy were done to differentiate between different lesions .Patients were treated according to underlying conditions and type of seizure . EEG was done routinely for all patients in the interictal period .

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2008).

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS AND ANALYSIS

Epidemiology

Majority of the patients were from in and around Madurai . Age of patients varied from 13 to 72 . Majority of patients were from low socioeconomic class .

Age distribution of patients is given in table 1.

Table 1 : Age distribution

AGE GROUP	CASES	
	No.	%
Upto 20 year	11	11
21-40 years	46	46
41 – 60 years	30	30
> 60 years	13	13
Total	100	100
Range Mean S.D.	13-72 years 39.4 years 16.6 years	

Table 2 : Sex distribution

Sex	CASES	
	No.	%
Male	58	58
Female	42	42
Total	100	100

Out of 100 cases 58 were males and 42 were females

Table 3 : Type of Seizure

Type of Seizure	CASES	
	No.	%
Focal	12	12
Focal Generalised	21	21
GTCS	63	63
EPC	4	4
Total	100	100

Out of 100 cases 12 patients presented with focal seizures , 21 with focal seizure with secondary generalization and 63 with GTCS .Only 4 patients were presented with epilepsia partialis continua . Out of these 100 cases 21 had Status epilepticus .Most common presentation was generalised tonic clonic siezures .

Table 4 : Type of Seizure and Age

Age group	% of Type of Seizure			
	Focal	Focal Generalised	GTCS	EPC
Upto 20 year	9.1	-	90.9	-
21-30 years	10.7	35.7	53.6	-
31 – 40 years	16.7	22.2	61.1	-
41 – 50 years	7.1	28.6	50	14.3
51-60 years	12.5	18.8	62.5	6.3
> 60 years	15.4	-	76.9	7.7
Mean S.D.	39.4 16.7	36.2 11.8	39.6 18	56.3 9.9

In all age groups the most common type of seizure was GTCS . Focal seizure was common in middle age . Patients presented with EPC was older adults or elderly .GTCS was most common in young patients .

Table 5 : Associated Symptoms

Symptoms	Absent		Present	
	No.	%	No.	%
Fever	88	88	12	12
Headache	69	69	31	31
Vomiting	69	69	31	31
Trauma	99	99	1	1
CSOM	97	97	3	3

Patients with acute symptomatic seizures due to infections and CVT had headache ,vomiting and fever . Only 12 patients had fever at the time of seizures. Patients with meningitis ,brain abscess ,encephalitis were presented with fever . Headache and vomiting was there for 31 patients . Most of these patients had mass lesions like tumor ,neurocysticercosis ,tuberculoma or abscess and CVT. History of CSOM was there for 3 patients with brain abscess and septic thrombosis of veins.

Table 6 Past Illness

Past illness	CASES	
	No.	%
None	69	69
DM	13	13
HTN	13	13
Tuberculosis	1	1
Meningitis	1	1
Malignancy	5	5
HIV Positive	2	2
Cardiac problem	2	2
CKD	4	4

Out of 100 patients 13 patients had history of diabetes and hypertension. History of treatment for malignancy of lung and breast was present for 5 patients . 2 patients presented with GTCS were HIV positive and one patient had history of tuberculosis . Twelve cases had more than one past illness.

Table7: Altered Sensorium

Altered Sensorium	CASES	
	No.	%
Absent	14	14
Drowsy	64	64
Stupor / Coma	22	22
Total	100	100

Normal level of conciousness was present only for 14 patients . All the other patients had some change in level of conciousness . Out of 100 patients 64 were drowsy and 22 patients were in stupor or coma . Ophthalmoscopic examination was normal in 64 patients and there was papilledema for 20 cases and retinopathy for 16 cases . Blood pressure was high in 23 patients of these 9 patients were in Pre- hypertension stage , 9 in stage 1 hypertension and 5 were in stage 2 hypertension.

Table 8 Focal Neurological Deficit

Focal Neurological Deficit	CASES	
	No.	%
Absent	70	70
Right hemiplegia	6	6
Left hemiplegia	17	17
Monoplegia – Right	1	1
Monoplegia – Left	3	3
Homonymous hemianopia	1	1
Cranial nerve Palsy	2	2
Total	100	100

Out of 100 patients with acute symptomatic seizures 30 patients had focal neurological deficit . Hemiplegia was there for 23 patients . Monoplegia was there for 4 patients . Hemianopia and cranial nerve palsy was present for 3 patients . All these patients weakness persisted for more than 5 days of hospital stay . None of these patients had todd's palsy .

All baseline investigations done for all patients . Out of 100 cases 8 patients had hyperglycemia and 2 had hypoglycemia . Both hypoglycemia and hyperglycemia resulted in seizures and controlled with correction of glycemic status . There was 11 patients with elevated urea and creatinine of these 8 had urea >100mg/dl , 3 had <100mg/dl, and creatinine >8mg /dl for 8 patients, <8mg /dl for 3 patients . All these patients had features of uremia and seizure controlled with AED followed by dialysis . Out of 100 cases 7 patients had hyponatremia with Na values <116 meq/l .Out of these 7 patients 3 had other associated metabolic abnormalities and 4 patients seizure was purely due to hyponatremia . Out of these 2 patients had hypothyroidism .

LFT was normal in all pateints . Chest X-ray was abnormal in 13 patients . Out of 13 pateints 4 had mass lesion , 5 had tuberculosis and 4 had cardiomegaly. ECG was normal in 82 patients ,left ventricular hypertrophy was there for 14 patients ,features of coronary artery disaese was there for 3patients and 1 patient had arrhythmia .

Table 9: EEG

EEG	CASES	
	No.	%
Normal	54	54
Diffuse slowing	10	10
Focal spikes & sharp waves	22	22
Bilateral spikes & waves	14	14

EEG was normal in 54 cases out of 100. Out of 46 abnormal cases 10 showed diffuse slowing , 22 cases showed focal spikes and sharp waves and 14 showed bilateral spikes and waves .

Table 10: Type of Seizure and EEG findings

EEG findings	% of Type of Seizure			
	Focal	Focal Generalised	GTCS	EPC
Normal	42	72	50	75
Abnormal	58	28	50	25

EPC – epilepsia partialis continua

GTCS- generalised tonic clonic seizures

EEG was abnormal in 50 % of patients with GTCS . In patients with focal siezure 58% of cases EEG was abnormal . In patients with secondary generalised seizure only 28 % showed abnormal recording and 72% were normal .

Out of 100 cases 46 cases showed abnormal record . CNS infections showed abnormal record 64 % of patients . Post traumatic seizures showed abnormal EEG 66% of patients .

TABLE 11:Underlying Cause and CT Scan findings

CT Scan findings	% of Underlying Cause						
	Infection	Metabolic	CVT	Stroke	Tumor	Calcified Granuloma	Others
Normal	3	100	12	-	-	-	33.3
Abnormal	97	-	88	100	100	100	66.7

CT brain was taken for all cases of acute symptomatic seizures . It was normal in 28% of cases . It was normal in all cases of metabolic causes of siezures. Showed abnormal report in all cases of stroke, tumor and calcification .In patients With CNS infections 97% showed abnormal report. In 3% of cases of acute symptomatic seizures CT was normal and MRI was abnormal . In case of CVT 88% of times it was abnormal .

Out of 100 patients 10 showed haemorrhage, 6 showed Infarct, 8 showed tuberculoma, 5 Neurocysticercosis, 5 Tumour primary, 4 Secondary, 6 Abscess, of the 6 cases of abscesses 3 showed CSOM with CVT ,7 Ring enhancing lesions, 11 Calcification, 11 CVT, 3 CSOM, Osteomyelitis of bone with cerebritis 1 case ,arachnoid cyst 1case and cerebral oedema was present in 9 cases .

MRI brain was taken for 43 patients . MRI was not taken for metabolic

cases and for patients whose CT brain is diagnostic . All the 43 patients CT was inconclusive and the diagnosis could not be made .There was 11 cases with tuberculoma , 5 cases of neurocysticercosis , 3cases of brain abscess , 4 cases of encephalitis and 1case of meningitis . Patient with mass in the CT brain 4 showed primary brain tumor and 2 showed mutiple secondaries . 2 patients had arteriovenous malformations in the brain . One of them had chronic infarct along with AVM .Out of 43 cases 11 cases of CVT was there . One case of hemangioma was present . 2 cases showed hemorrhage .

CT brain was normal in 3 patients and MRI was abnormal in these 3 cases one had CVT , one had arteriovenous malformations and another one had encephalitis .

Table 12: Underlying Cause

Underlying Cause	CASES	
	No.	%
Infection	33	33
Metabolic	25	25
Stroke	11	11
Calcified Granuloma	10	10
CVT	9	9
Tumor	9	9
Others (Cyst,AVM etc)	3	3

Out of 100 cases 33% of cases were due to infection ,25% metabolic and toxic causes, 11% stroke , 9% cases tumor , 9 cases were CVT , 10 cases were single calcified lesions . 3% were classified as others 1 arachnoid cyst , 1 AVM, 1 Alzheimers disease . Out of 33 cases of infections 3 had septic thrombosis of sinuses with brain abscess were there .

Table 13: Underlying Cause and Age

Age group	% of underlying Cause						
	Infection	Metabolic	CVT	Stroke	Tumor	Calcified Granuloma	Others
Upto 20yrs	63.6	9.1	9.1	9.1	-	-	9.1
21-30 yrs	32.1	14.3	21.4	-	7.1	21.4	3.6
31 – 40 yrs	44.4	11.1	5.6	16.7	5.6	16.7	-
41 – 50 yrs	42.9	28.6	7.1	7.1	7.1	-	7.1
51-60 yrs	18.8	43.8	-	12.5	18.8	6.3	-
> 60 years	-	53.8	-	23.1	15.4	-	7.7
Mean S.D.	32.9 12.3	49.6 16.5	26.7 9.3	48.3 18.2	48.3 16.5	30.6 8.8	45 21.7

Patients upto 20 years of age most common cause was infections . Young adults from 20-30years infection was most common followed by CVT and calcified granuloma . In patients with 30-40 age group most common cause was infection followed by stroke and calcified granuloma . Patients aged 41-50 infection was followed by metabolic causes . Patients aged 51-60 most common etiology was metabolic abnormalities and next common was tumor

and infections . Patients >60 years metabolic causes were leading followed by stroke and tumors .

Infections were common in younger patients ,metabolic abnormalities were the common causes in older adults and elderly . CVT and single calcified lesion was common in 20-30 years of age . Stroke and tumors common in patients >50 years of age .

FIGURE 14:CAUSE & SEX DISTRIBUTION

CAUSE	MALE	FEMALE
Infection	64	36
Metabolic	60	40
CVT	34	66
Stroke	60	40
Tumor	55	45
Calcified Granuloma	60	40
Others	66	34

Most of the causes were common in males than females except CVT. It was common in females . 66%of CVT occurred in females .

Table 15: CHARACTERISTICS OF THE 21 CASES OF STATUS EPILEPTICUS

Underlying cause	Frequency	Percent
Infection	8	38.1%
Metabolic	3	14.3%
CVT	6	28.6%
Stroke	2	9.5%
Tumor	1	4.8%
Calcified Granuloma	1	4.8%
Total	21	100.0%

Status epilepticus was the presentation in 21 cases .Most common cause of status epilepticus was CNS infections followed by CVT . 3 patients with status epilepticus there was metabolic causes . Out of 21 cases there were 14 males and 7 females.

TABLE 16:ETIOLOGY OF ACUTE SYPTOMATIC SIEZURES

INFECTION -MOST COMMON CAUSE

Diagnosis	Frequency	Percent
Tuberculoma	13	39%
Neurocysticercosis	7	21%
Encephalitis	5	15%
Brain abscess	3	9%
Brain abscess with CVT	3	9%
Osteomyelitis,epidural abscess,cerebritis	1	3%
TB meningitis	1	3%
Total	33	100.0%

Out of 33 cases of infections 13 cases (40%) were due to tuberculoma , 7 cases were due to neurocysticercosis (21%) , 5 cases of encephalitis (15%) , 3 cases of brain abscess and 3 cases of brain abscess with CVT secondary to CSOM was there. One case of osteomyelitis of parietal bone and epidural abscess along with cerebritis was there.

TABLE 17: METABOLIC -SECOND MOST COMMON CAUSE

Diagnosis	Frequency	Percent
CKD Uremia	9	36%
Hyperglycemia	4	16%
Hypoglycemia	2	8%
Hyponatremia	4	16%
Toxins	6	24%
Alcohol withdrawal	2	
Cypermethrin poisoning	2	
OPC poisoning	2	
Total	25	

Out of 25 cases of metabolic cases there were 9 cases of uremia . 4 patients had hyperglycemic hyperosmolar state, 4 had hypontremia and 2 patients had hypoglycemia .There were 6 cases of seizures due to toxins of these 2 were due to organophosphorus compound poisoning (OPC) , another 2cases due to cypermethrin poisoning . There was 2 patients with seizures due to alcohol withdrawal.

Out of 100 cases 11 cases were related to stroke , of these 6 cases(54%) were due to hemorrhagic stroke , 5 were(46%) related to infarction . Out of

6 patients with hemorrhagic stroke one was due to rupture of hemangioma,one was due to thrombocytopenia related intracerebral hemorrhage and 4 cases were due to hypertensive intracerebral hemorrhage . Out of 5 cases of infarction related seizures , 3 were due to old infarct and 2 were due to acute infarct .One was secondary to AVM and one was cardioembolic stroke in rheumatic heart disease patient .

Out of 100 cases there were 9 cases of CVT and 3 cases of CVT with brain abscess secondary to CSOM. Of these 4 were males(34%) and 8 were females(66%) . Out of 8 females 4 were postpartum CVT (34%).

There was 10 cases of single calcified granuloma out of 100 patients of acute symptomatic seizures . It was most common in patients from 30 to 40 years of age . One patient with single calcified granuloma had status epilepticus.

There were 9 cases of tumors as a cause of acute symptomatic seizures. Of these 4 were primary brain tumors and 5 were secondary tumors . Of these secondary tumors ,3 had carcinoma lung and 2 had carcinoma breast . Out of 100 cases ,4 cases classified as others ,out of that 1 had arachnoid cyst ,1 had alzheimers disease and 2 had arteriovenous malformations.

DISCUSSION

Government Rajaji Hospital, Madurai is the only tertiary referral care hospital located in Madurai district. Various cases have been referred from Government sector hospitals like PHCs, Taluk hospitals, District head quarters hospitals, ESI hospital and many private hospital of not only from Madurai but also from near by districts. Study of acute symptomatic seizure patients from June 2008 to June 2009 included 100 patients.

DEMOGRAPHY

Acute symptomatic seizure is an important cause of morbidity in our part of the country . It is very important to find out the underlying cause and its treatment for prevention of reccurent seizure . Acute symptomatic seizure is different from epileptic syndromes as it is curable by treating the underlying cause . Even if AEDs are used to suppress recurrence of seizures, they generally do not need to be continued after the patient has recovered form the primary illness. These concepts are based on the basic assumption that acute symptomatic seizures presumably cease with the resolution of the precipitating cause or illness. But seizure can recur in conditions like tumor ,stroke and head injury .

There are 58 cases of males and 42 cases of females in our study . The mean age is 40 and there is patients from 13 to 72 years of age.Study by Sander et al [13] the proportions of males and females were similar. Usha Kant Misra et al ‘s study the median age of the patients was 37 years from 16-78

years. According to Jaishree T Narayanan et al the mean age of patients with acute symptomatic seizures was 49.07+20.29 years (six months to 80years) as they had included pediatric patients in their study .In our study 76% of patients are in the age group of 40 to 60 years ,11%of patients are less than 20 years and 13% are more than 60 years . Study by Sander et al 25% (21-28%) were younger than 15 years and 24% (21-28%) were 60 years or older. Twenty-four (36%) were aged 60 years and above in Jaishree T Narayanan et al study .

MODE OF PRESENTATION

In our study 63% of patients the type of seizure is GTCS and 21% focal with secondary generalisation and12% presented with focal seizure and 4% with epilepsia partialis continua . Our study correlates with previous studies. Study by Usha Kant Mishra et al generalized tonic-clonic seizure was the seizure type in 36 (55%) patients and in the remaining 30 (45%) patients, the seizure type was partial with or without secondary generalization. Of the 30 patients with partial seizures, 28 (93%) had complex partial seizure and two(7%) had epilepsia partialis continua in their study .According to Sridharan et al in the new cases of epilepsy 50% have seizures of partial origin and 50% of generalized origin before the age of 40 years . After 40 years, the proportion of partial epilepsy rises to 75% by the age of 75. Study by Clifford Schold a total of 56% of the patients had focal motor seizures, and in 44%, the seizures were generalized. Study by J. M. K. Murthy & Ravi Yangala type of

seizure was simple partial or complex partial with or without secondary generalization in 412 (78%) patients and either unlocalized or generalized in 114 (22%) patients.

Status epilepticus is present in 21% our patients . Most common cause for status epilepticus is infection(38%) followed by CVT (28%) and metabolic disturbances(14%). According to Jaishree T Narayanan et al 10 (15%) patients had seizure clusters and four (6%) patients presented with SE. Usha Kant Misra et al 's study 35 patients had convulsive status epilepticus, and 2 patients had nonconvulsive status epilepticus .

ETIOLOGY

Most common cause of siezure in our study is infections of CNS followed by metabolic and toxic causes . Infection is the cause of siezure in 33% of patients .Our study correlate with other studies from south india [3,4,5,6]. Out of 100 cases 33% of cases are due to infection , 25% due to metabolic and toxic causes, 11% due to stroke , 9% due to tumor , 9% due to CVT and 10% are due to single calcified lesions . 3% are classified as others include 1arachnoid cyst , 1 AVM, 1 Alzheimers disease .Out of 33 cases of infections 3 shows septic thrombosis of sinuses with brain abscess.

In our study 33% are due to infection and 10% are due to single calcified lesion . Another study from south India by Jaishree T Narayanan (6) showed central nervous system (CNS) infections in 32% patients. Study by J. M. K.

Murthy et al seizure occurred in close temporal association with an acute central nervous system (CNS) insult in 53% of patients. Infections of CNS including single CT enhancing lesion accounted for 77% of patients with acute symptomatic epilepsy. Study by Ravindra Kumar Garg et al[23] infective pathologies were the most common etiology.

In our study out of 33 cases of infections 13 cases (40%) are due to tuberculoma , 7 cases are due to neurocysticercosis (21%) , 5 cases of encephalitis (15%) , 3cases of brain abscess and 3 cases of brain abscess with CVT secondary to CSOM are there. One case of osteomyelitis of parietal bone and epidural abscess along with cerebritis is there. The distribution of the pathology according to Jaishree T Narayanan et al [6] in patients with CNS infections was meningoencephalitis in 43% and parenchymal granuloma in 57% of patients out of that 75% due to degenerative phase solitary cystic granuloma and 25% due to tuberculoma . Study by J. M. K. Murthy et al Neurocysticercosis, SCTEL and small single cerebral calcific CT lesion (SSCCCTL) together accounted for 40% of etiological factors and neurotuberculosis for 10%.

In our study neurotuberculosis accounts for 40% of infections and according to Jaishree T Narayanan et al it was 25% and J. M. K. Murthy et al 10%. Study by Ravindra Kumar Garg et al[23] tuberculosis was the commonest infective pathology. Usha Kant et al 's study there was 5 cases of tuberculous meningitis and 5 cases of tuberculoma brain was there . Brain

tuberculomas make up 5 to 8 per cent of intracranial masses in person in developing countries[18]. Before effective chemotherapy was available for tuberculosis, tuberculoma made up 20 per cent of intracranial lesions in one large series([19]).

In our study 21% of infections are due to neurocysticercosis and 10 cases due to small single cerebral calcific CT lesion . According to Jaishree T Narayanan et al parenchymal granuloma in 57% patients with CNS infections ,out of that 75% due to degenerative phase solitary cystic granuloma . Study by J. M. K. Murthy et al neurocysticercosis, SCTEL and small single cerebral calcific CT lesion (SSCCCTL) together accounted for 40% of etiological factors. Study by Ravindra Kumar Garg et al[23] second most common infection was neurocysticercosis following tuberculosis . According to Thussu et al [65] solitary cysticercus granuloma, a benign form of parenchymal neurocysticercosis, is considered to be the most common aetiology for SSECTL . According to Macro et al [64] neurocysticercosis is the most common parasitic disease of the CNS in developing countries . According to Montano et al an important proportion of seizure cases are associated with neurocysticercosis in endemic areas .

In our study 25% are due to metabolic and toxic causes . Jaishree T Narayanan [6] et al study showed metabolic disorders in 32% of cases of seizures . In our study there are 9 cases of uremia , 4 cases of hyperglycemic hyperosmolar state, 4 cases of hypontremia and 2 cases of hypoglycemia .

Study by Jaishree T Narayanan [6] out of 21 cases 15 were due to hyponatremia, 4were due to hyperglycemia, 2 were due to hypoglycemia . In our study, 6 cases of seizures are related to toxins, out of that , 2 are due to organophosphorus poisoning , another 2 cases cypermethrine poisoning and 2 cases are alcohol withdrawal. Study by Jaishree T Narayanan [6] there was 6 cases of alcohol related seizures and 2 cases of hypoxic encephalopathy .

In our study 11% of cases the cause of seizure is stroke and 10% due to CVT . Out of these 6 cases(54%) are due to hemorrhagic stroke , 5 are (46%) related to infarction. By Jaishree T Narayanan [6] cerebrovascular diseases (ischemic, venous and hemorrhagic) in 21% cases and by JMK Murthy vascular in 14% (Ischemic 6%,Haemorrhagic 5% , CVT 3%) of cases .Mean age of patient presenting with seizure due to stroke was 48+/- 18 years . Studies from developed countries acute symptomatic seizure due vascular causes were common . Study by Sander et al[13] vascular disease in 15% (12-18%) , among older subjects 49% (41-58%) were due to vascular disease. Study by J. M. K. Murthy et al cerebrovascular diseases were the risk factors in 48% of patients with remote symptomatic epilepsy and cerebrovascular diseases were the etiological factors in 64% of patients aged >40 years . By Ettinger AB et al the most common single cause of seizures was infarction or hemorrhage (54%). According to Sridharan et al cerebrovascular disease is the most commonly identified cause among adults,37% of symptomatic seizures .

There are 9 cases of CVT and 3 cases of CVT with brain abscess secondary to CSOM in our study . Out of these 4 patients are males (34%) and 8 patients are females(66%) . Out of 8 females 4 are postpartum CVT (34%). CVT is a common cause of seizure in the postpartum period in our hospital . Jaishree T Narayanan et al study 3% of cases were due to CVT . Study by Dr J. M. Murthy, Department of Neurology, Nizam's Institute of Medical Sciences , Hyderabad 3% of cases were due to CVT . Cortical sinovenous thrombosis is an important cause of acute symptomatic seizures among young patients with cerebrovascular diseases . A number of conditions have been etiologically linked to cortical sino venous thrombosis. But in India the majority of cases are related to pregnancy and purperium.

Seizure associated with fever ,headache or vomiting should be investigated to find out the underlying cause . In our study patients with infections and CVT had headache ,vomiting and fever . Only 12 patients had fever at the time of seizures. Patients with meningitis , brain abscess, encephalitis were presented with fever . Headache and vomiting was present for 31 patients . Most of these patients had mass lesions like tumor neurocysticercosis ,tuberculoma abscess or CVT. In our study 30 patients had nuerological deficit , most of the cases due to CVT , tumor or stroke .Cerebral AVM and arachnoid cyst are other rare causes for seizure . For the diagnosis of AVM , MRI brain is the investigation of choice .

Immediate non-contrast CT is useful for emergency patients presenting with seizure to guide appropriate acute management, especially where there is an abnormal neurologic examination, predisposing history, or focal seizure onset. MRI has been shown to be highly sensitive and specific in identifying the underlying pathology in partial epilepsy. MRI may determine patient selection for surgery and directly affects the presurgical evaluation and operative strategy. Therefore MRI should be performed early to avoid unnecessary medication in patients with resectable intracranial mass lesions.

In our study out of 100 patients 46 showed abnormal EEG , 21% showed diffuse slowing ,47%cases showed focalspikes and sharp waves and 30% showed bilateral spike and wave patern . The frequency of various ictal discharges was variable in different studies. In the series by Granner and Lee ictal discharges were generalized in 69%, diffuse with focal predominance in 18%, and focal in 11%. In another study the discharges were generalized in 59% and lateralized or localized in 41%.In our study EEG was abnormal in patients with tumor , tuberculoma neurocysticercosis and in a few cases of CVT and calcification .

Seizures may herald or complicate acute neurological and medical disorders.The etiological spectrum in the present study was distinctly different when compared to the data from developed countries and it well correlate with other studies from developing countries and other studies from south India. New-onset acute symptomatic seizures are different from unprovoked seizures

in that they generally do not recur and usually do not need long-term AED therapy.

However, this study suggests that the risk of seizure recurrence or SE after the first seizure is likely to be high in patients with acute focal cerebral lesions and diffuse CNS infections like meningoencephalitis and encephalitis. Of the patients who had seizure recurrence or developed SE, in 38% the pathology was infection-related and the other commonality was cortical gray matter involvement. Probably this group of patients, particularly patients with CNS infections, with high risk of seizure recurrence may need AED prophylaxis, at least for the period of resolution or stabilization of acute CNS insult .

When considering the results of this study the limitations of the study must be recognized. This is a highly selected population and the findings may not be generalizable. In developing countries CNS infections like Japanese encephalitis, tuberculous meningitis, bacterial meningitis and NCC are endemic and are frequent risk factors for new-onset acute symptomatic seizures. There is a need to study a large population of patients with these pathologies for the risk of recurrence of seizures as it may have therapeutic implications, possible AED prophylaxis.

SUMMARY

The study "Demographic, clinical, investigational and etiological profile of acute symptomatic seizures" was a cross sectional study of 100 patients admitted with first episode seizures in Government Rajaji Hospital Madurai. Patients who satisfied the inclusion criteria were included in the study and a detailed history was taken from the relatives about the type of seizure and comorbid conditions. Examination of CNS was done to find out any under lying neurological deficit including fundus examination to look for papilledema. Investigations done in all patients were blood sugar, urea, creatinine, serum electrolytes and liver function test. ECG, chest x-ray EEG and CT brain was done for all patients. MRI brain was done for indicated cases. Analysis of the type of seizure, etiology of seizure and specific pathology was done. This study shows that CNS infections like tuberculoma, neurocysticercosis, encephalitis and brain abscess are treatable causes of seizures in our hospital. So identification of the underlying pathology by various investigations and neuroimaging modalities are very important in the management of these patients. Patients with metabolic abnormalities, on correction of the underlying condition seizure will be controlled and diagnosis is important to avoid long term AED treatment.

CONCLUSION

1. Acute symptomatic seizure can develop at any age and it is most common in patients 20-40 years of age .

2. Most common cause of acute symptomatic seizure is CNS infections in our patients . Second most common cause is metabolic abnormalities and following that CVT ,stroke and tumors of brain both primary and secondary .

3. Infections and single calcified lesions are common in young patients and metabolic conditions are common in older patients .

4. CVT is an important cause of seizure in females especially during peripartum period . In males CVT is secondary to infection or hypercoagulable conditions .

5. Tuberculoma brain and neurocysticercosis are the most common CNS infections causing seizures followed by brain abscess and encephalitis .

6. Metabolic causes of seizures are most common with uremia followed by toxins and other cases are due to hyponatremia, hyperglycemia and hypoglycemia .

7. Immediate non-contrast CT is useful for emergency patients presenting with seizure to guide appropriate acute management, especially where there is an abnormal neurologic examination, predisposing history, or focal seizure onset.

8. MRI has been shown to be highly sensitive and specific in identifying the underlying pathology in partial epilepsy.

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PROFORMA

Name:

Age:

Sex:

Residence:

socioeconomic status

education:

occupation

income:

COMPLAINTS:

Type of seizure:

Status epilepticus:

H/O fever

H/O headache:

H/O vomiting:

H/O loss of consciousness:

H/O weakness:

H/O exanthematous illness:

H/O joint pain/ rash

H/O diarrhea

Post partum state

H/O trauma

H/O CSOM

PAST ILLNESS

Tuberculosis

Diabetes

Hypertension

Meningitis

Malignancy/ R_x for malignancy

PERSONAL HISTORY

pork eating

level of sanitation

contact with PT

H/O abortions

tobacco use

Alcohol use

Family H/O seizure:

EXAMINATION

Anemia

Cyanosis PR:

Lymphadenopathy BP:

CNS:

HF:

Cranial nerves:

Fundus:

Focal deficit:

Other systems:

INVESTIGATIONS:

Hb	TC	DC	ESR
Sugar	urea	creatinine	
Na	K	Cl	HCO3
LFT Bilirubin total	direct	indirect	
AST	ALT	ALP	
ECG		EEG	
Carotid Doppler		CSF:	
CT/MRI			

DIAGNOSIS:

KEY TO MASTER CHART

Seizures 1- focal 2- focal – generalized

3- GTCS 4- EPC 5- Status epilepticus

Fever 1 – absent, 2- present

Headache 1 – absent 2- present

Vomiting 1 – absent 2- present

Altered sensorium 1 – absent 2- drowsy 3- stupor / coma

Focal neurological deficit

1 –absent 2- Rt hemiplegia 3-Lt hemiplegia 4- Monoplegia Rt

5- monoplegia Lt 6- Homonymous hemianopia 7-CN palsy

Puerperal state

1- Puerperal 2- Non puerperal 3- Not applicable

Trauma 1-absent 2-present

CSOM 1-absent 2-present

Past illness 1-none 2-DM 3-HTN 4-Tuberculosis 5-meningitis

6-malignancy 7-HIV positive 8-IHD/RHD 9-CKD

Personal history 0-none 1-tobacco use 2-alcohol

3-contact with tuberculosis 4-poor sanitation

Blood pressure 1-normal 2-preHTN 3-stage 1 3-stage 2

Fundus 1-normal 2-Retinopathy 3- Papilledema

Hb(gms) 1- ≤ 5 gms 2- 5-8 gms 3- > 8 gms 4->10gms

Blood sugar 1-normal 2-high 3-Low

Blood urea 1-normal 2-<100 mg/dl 3->100mg/dl

Creatinine 1- normal 2-<8mg/dl 3->8mg/dl

S.Electrolytes 1- normal 2-hyponatremia

LFT 1- normal 2-abnormal

Chest xray 1-normal 2-mass 3-tuberculosis 4-cardiomegaly

ECG 1- normal 2-LVH 3-CAD 4-Arrythmia

EEG 1- normal 2-diffuse slowing 3-focal spikes and sharp waves
4-bilateral spikes and sharp waves

CT scan 1- normal 2-heamorhage 3-infarct
4-tuberculoma 5-neurocysticercosis 6-secondary
7-abcess 8-CVT 9-arachnoid cyst
10- osteomyelitis of bone with cerebritis 11-CSOM
12-ring enhancing lesion 13-tumour primary
14-calcification 15-Oedema 16-Atrophic brain

MRI 1-not done 2- tuberculoma 3- neurocysticercosis
4- tumour primary 5- secondary 6- CVT
7-AVM 8-encephalitis 9-heamangioma
10- abscess 11-Heamorrhage 12-meningitis

Figure 1:Age distribution

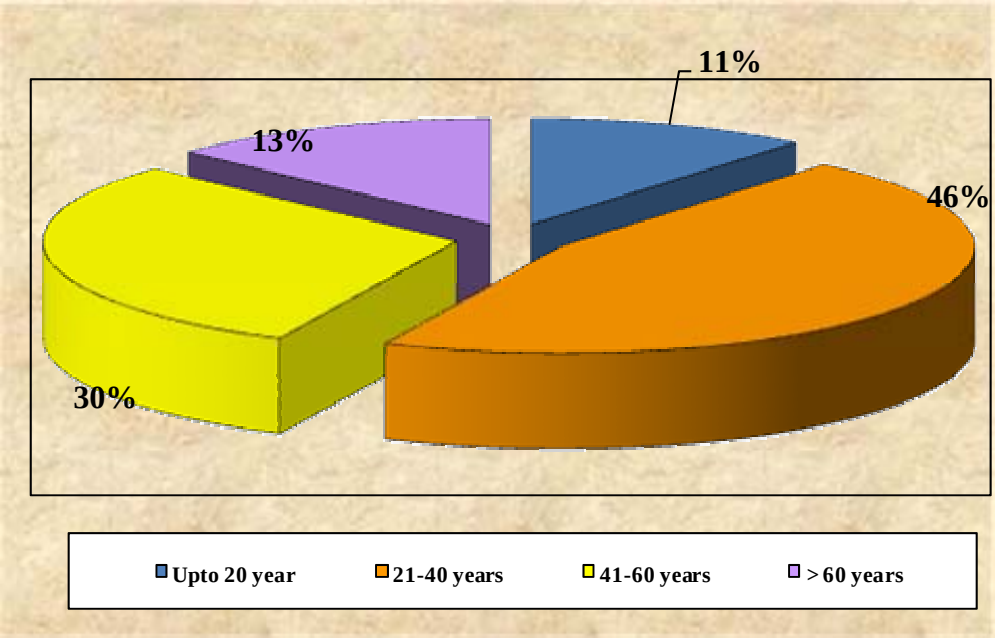


Figure 2: sex distribution

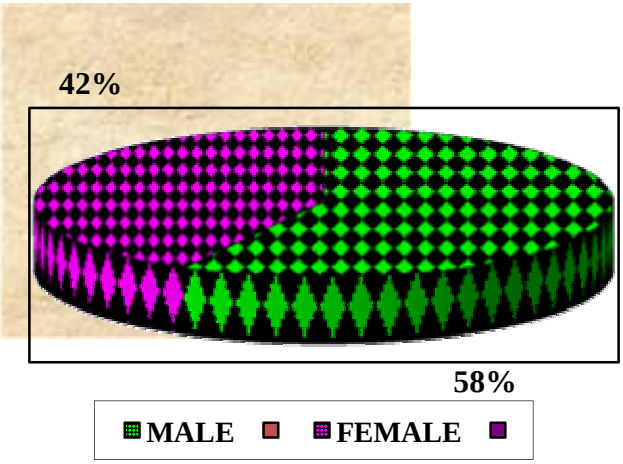


Figure 3: TYPE OF SEIZURE

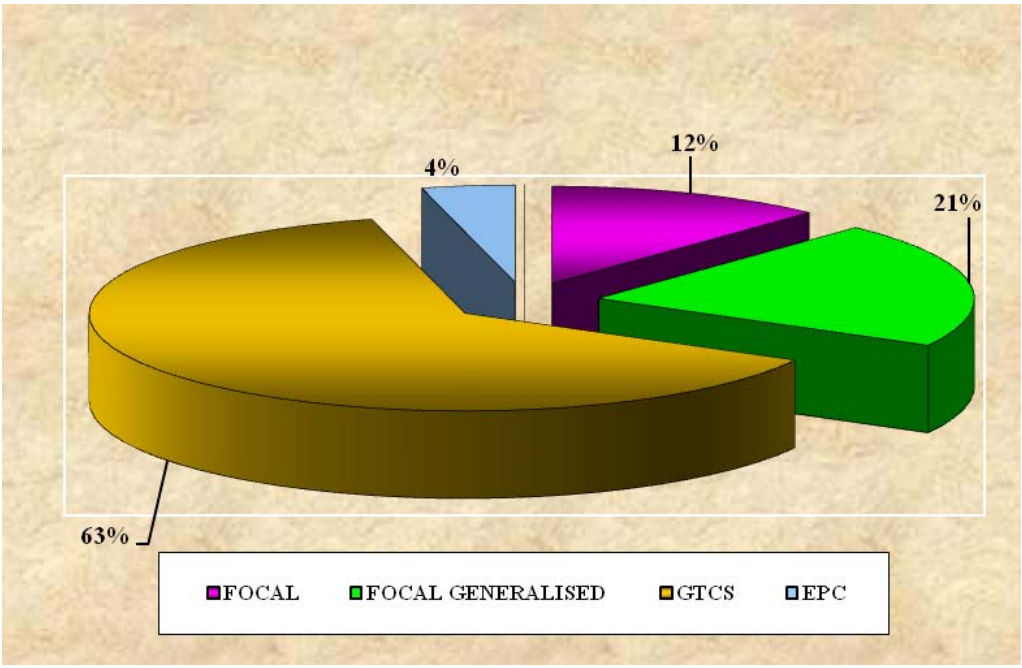
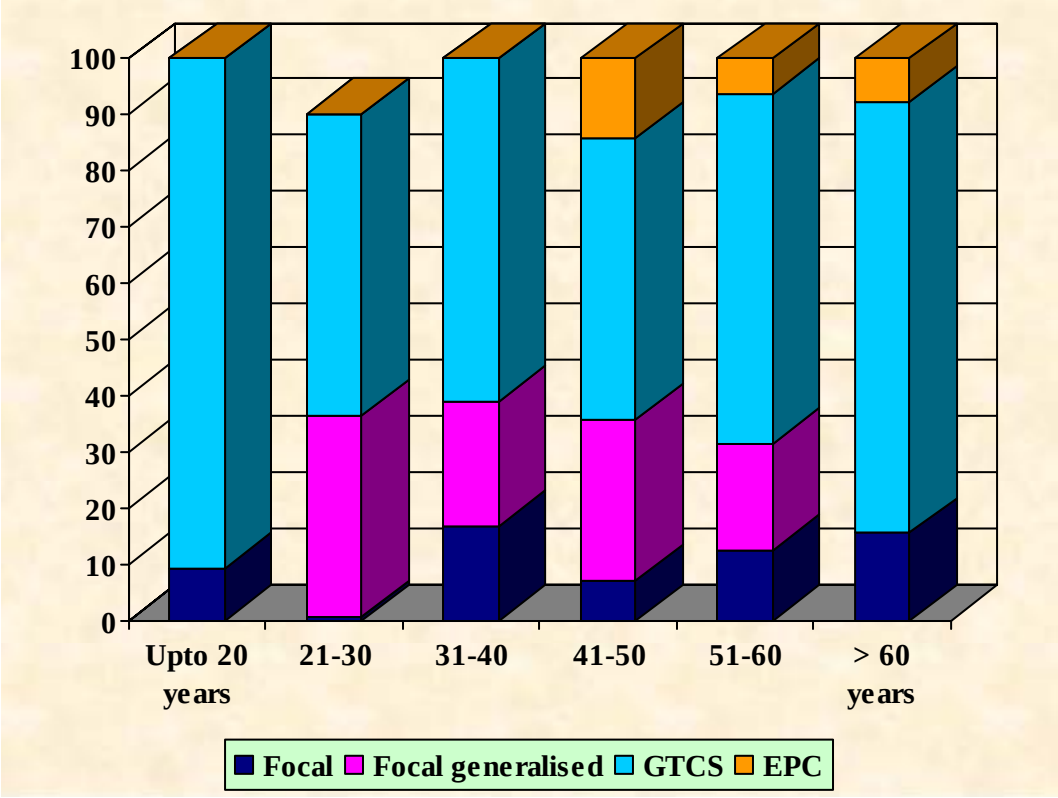
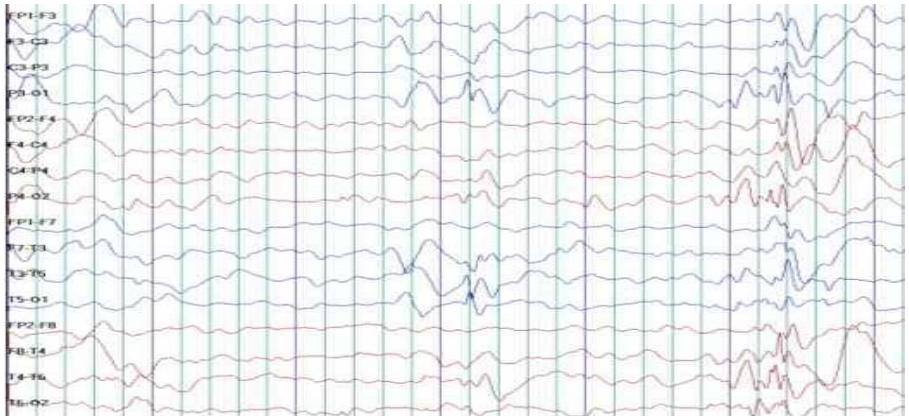


FIGURE 4 : TYPE OF SEIZURE AND AGE



EEG with MULTIFOCAL AND POLYSPIKE –WAVE PATERN



EEG-DORSOLATERAL FRONTAL SPIKES

EEG SHOWING GENERALISED ATYPICAL SPIKE WAVE DISCHARGES

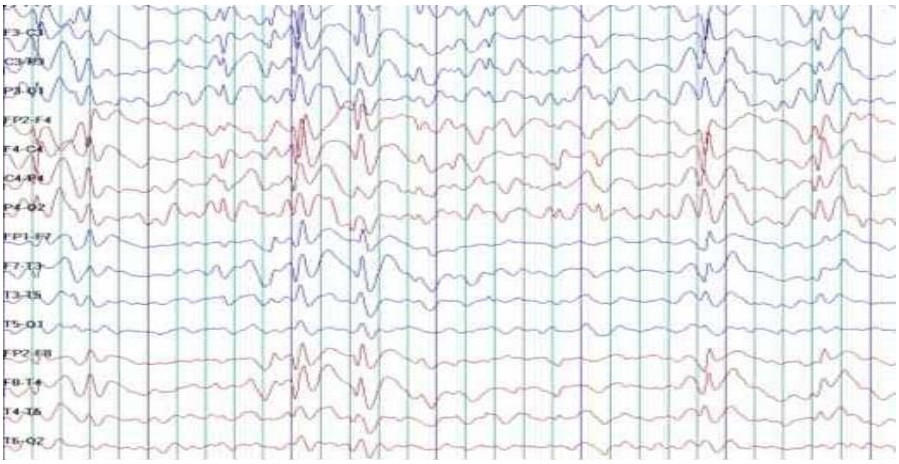


FIGURE 5 : EEG FINDINGS AND UNDERLYING CAUSE

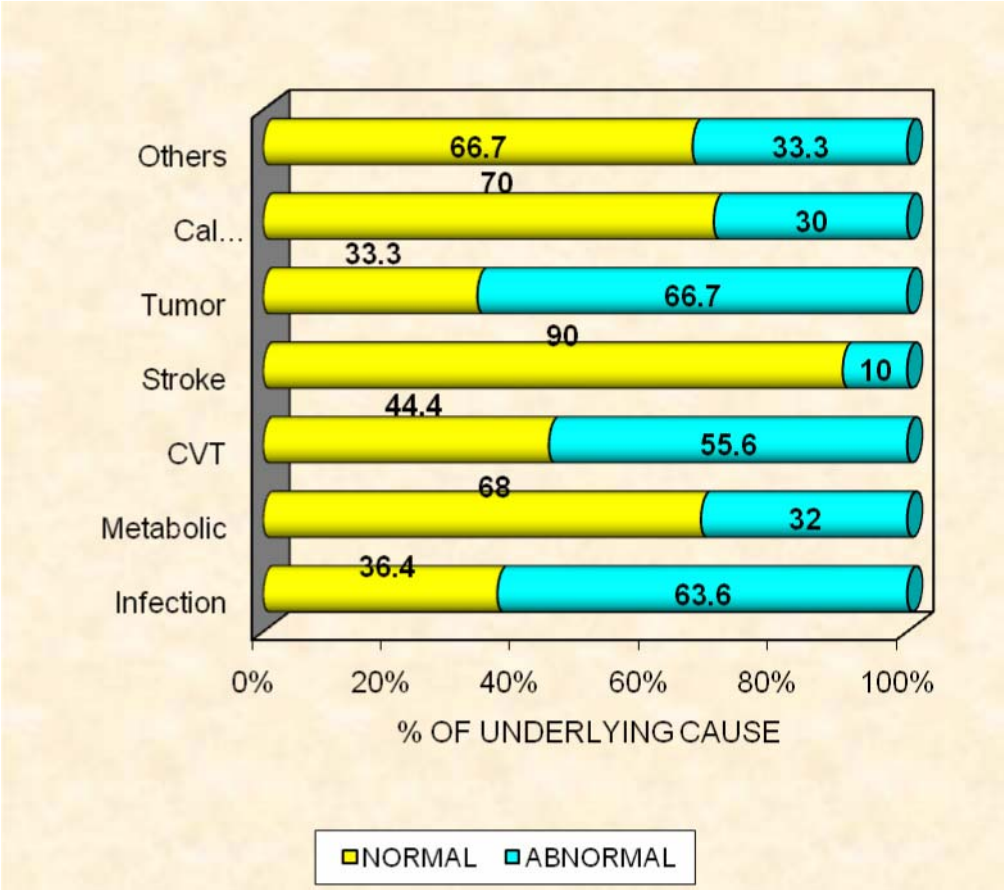
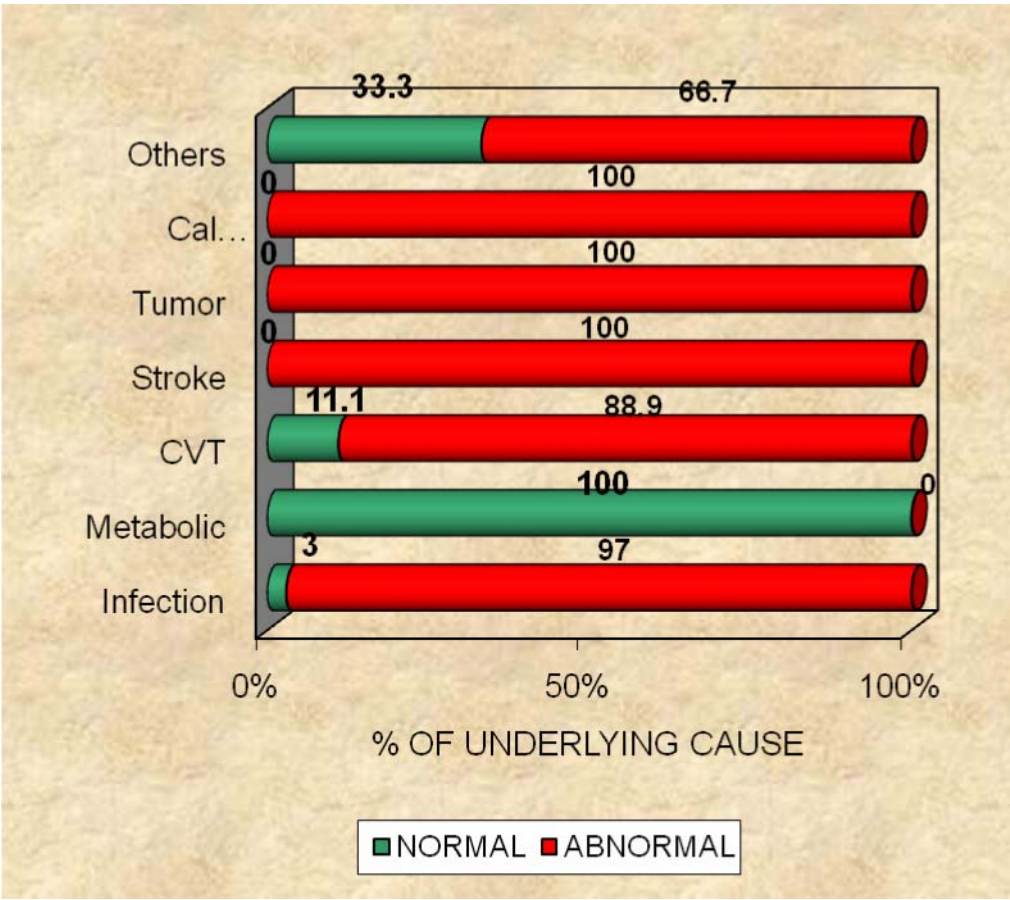
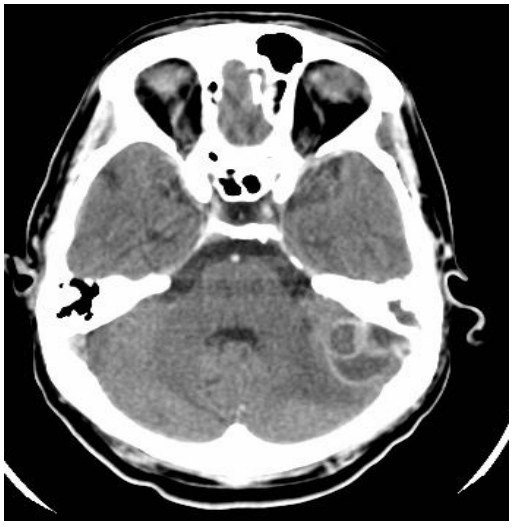


FIGURE 6: CT SCAN FINDINGS AND UNDERLYING CAUSE



LEFT FRONTAL LOBE NEUROCYSTICERCOSIS , CALCIFIED LESION PARIETAL LOBE 	**RING ENHANCING LESION - TUBERCULOMA** 
CEREBELLAR ABCESS SECONDARY TO CSOM 	**MULTICENTRIC GLIOBLASTOMA MULTIFORME** 

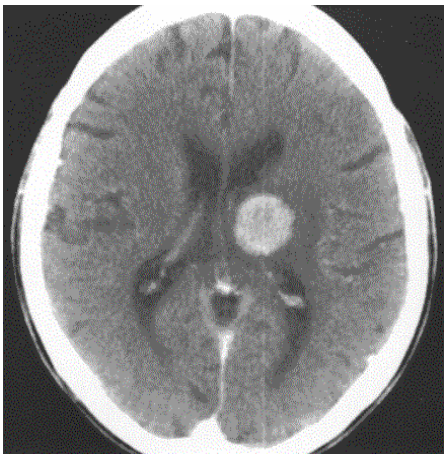
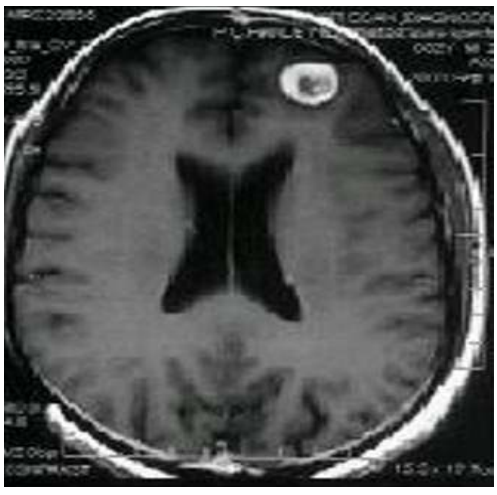
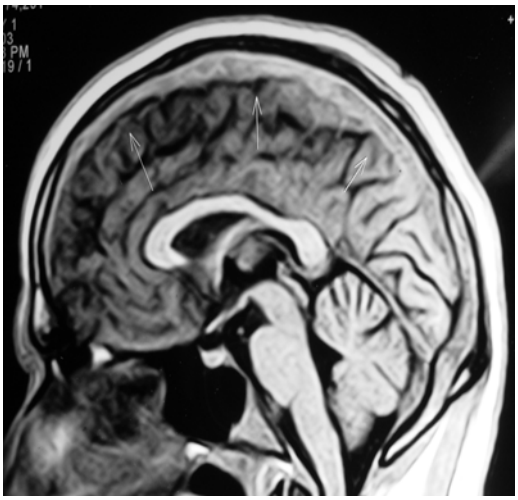
INTRACEREBRAL HEMORHAGE WITH INTRAVENTRICULAR EXTENSION 	**PRIMARY BRAIN TUMOR** 
MRI BRAIN NUEROCYSTICERCOSIS WITH SCOLEX 	**MRI- MID SAGITTAL SECTION SHOWING EVIDENCE OF SUPERIOR SAGITTAL SINUS THROMBOSIS** 

FIGURE 7: UNDERLYING CAUSES

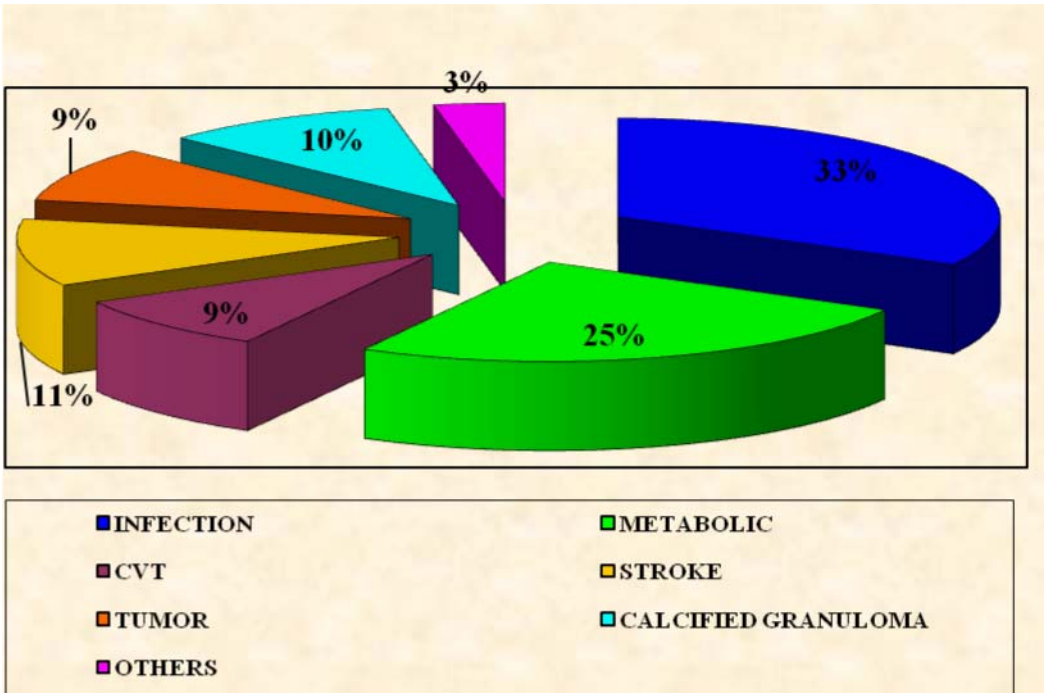


FIGURE 8: UNDERLYING CAUSE AND AGE

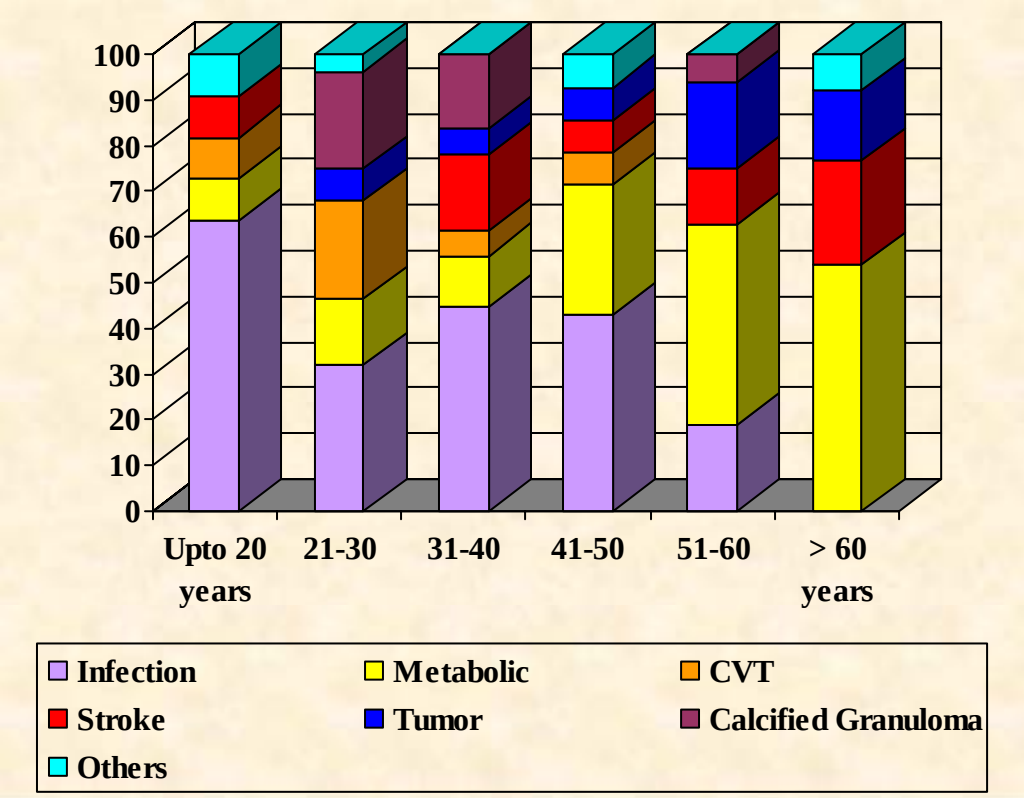


FIGURE 9: CAUSE & SEX DISTRIBUTION

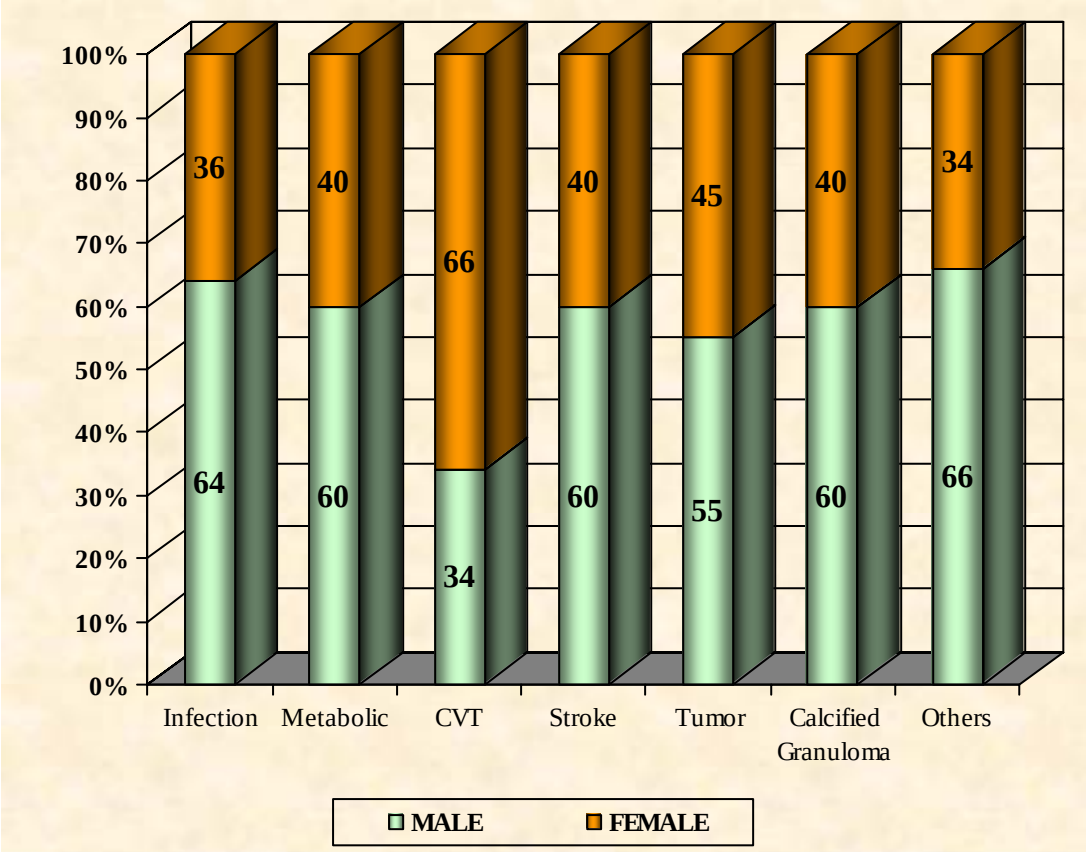


FIGURE 10 : ETIOLOGY : INFECTION

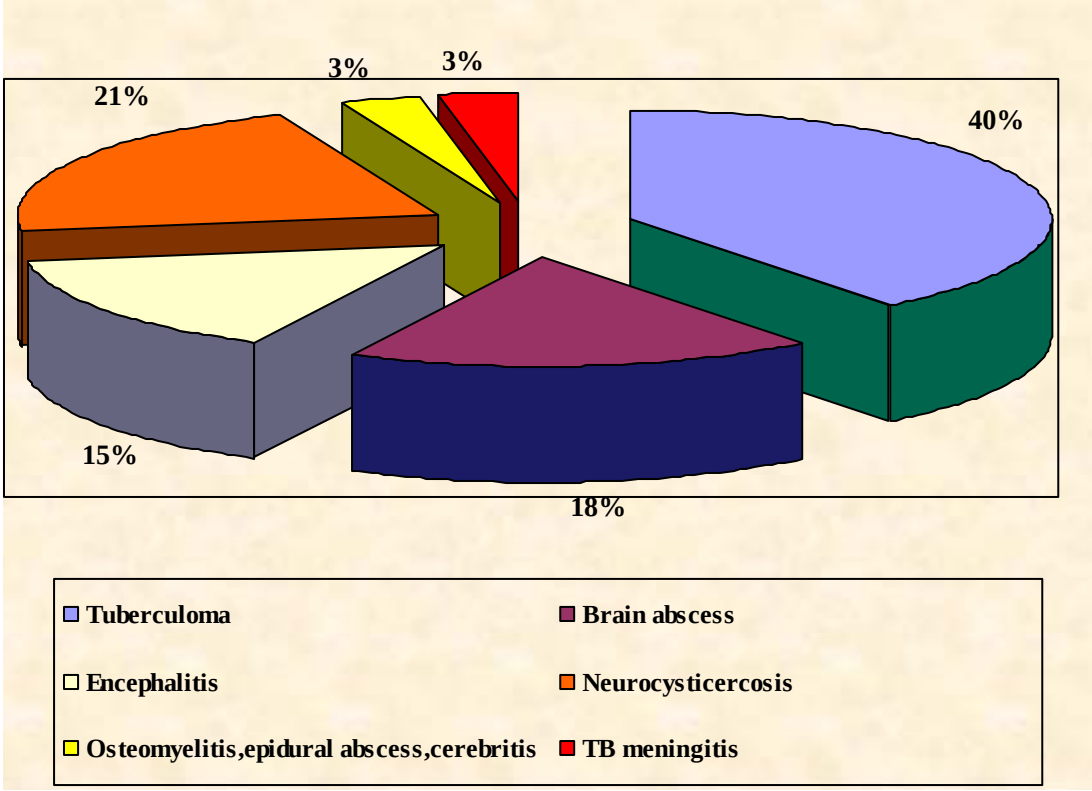
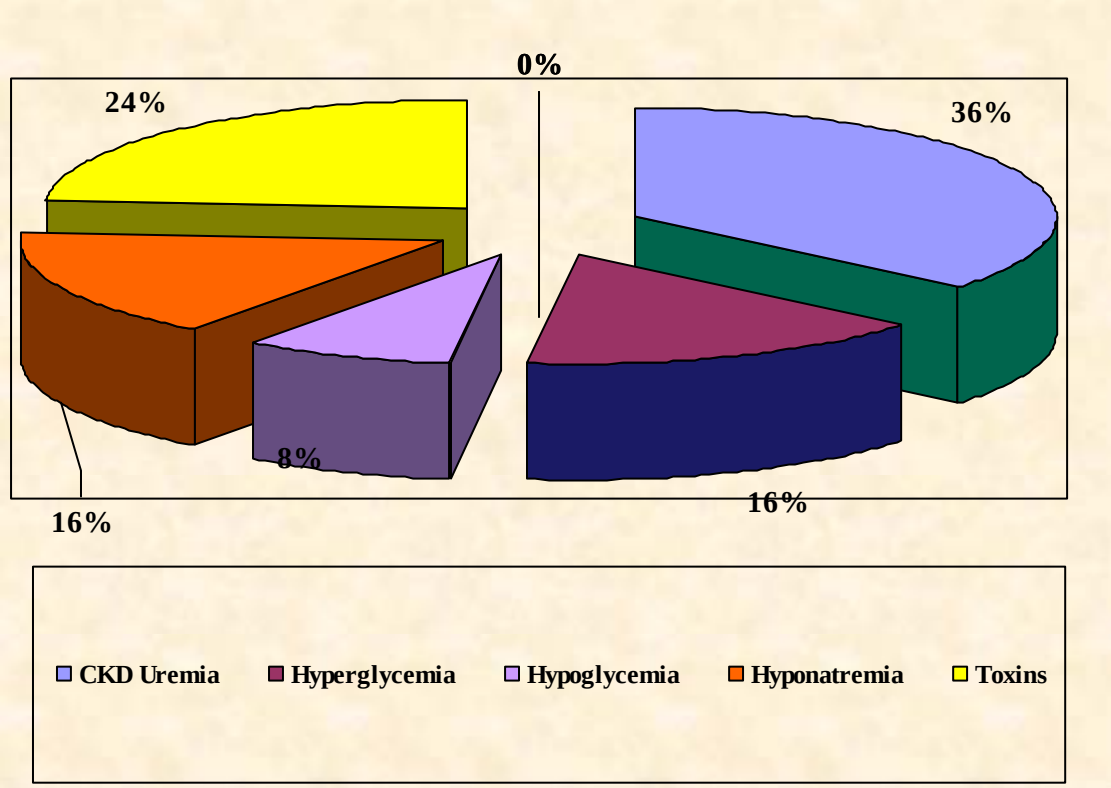


FIGURE 11 : ETIOLOGY : METABOLIC AND TOXIC CAUSES



S.No	Name	Age	Sex	Type of seizure	fever	Headache	Vomiting	Altered sensorium	FND	Puerperal state	Trauma	CSOM	Personal history	Past illness	Fundus	Blood pressure	Hb	Sugar	Blood urea	Creatinine	Electrolytes	LFT	Chest xray	ECG	EEG	CT Brain	MRI Brain	Diagnosis	
1	Ramallingam	45	M	3,5	1	1	1	2	1	3	1	1	0,4	3	1	4	4	1	1	1	1	1	1	2	2	8,2	6	CVT,SSS thrombosis,Lt hemiplegia	CVT
2	Arumugam	50	M	3	1	1	1	1	2	3	1	1	1,4	1	1	1	4	1	1	1	1	1	1	1	1	2	1	Heamorhagic stroke	STROKE
3	Muthusamy	26	M	1	1	2	2	2	1	3	1	1	1,4	1	1	1	4	1	1	1	1	1	1	1	4	4	1	Tuberculoma multiple	INFECTION
4	Gurunathan	17	M	3,5	2	2	2	3	1	3	1	1	0	1	1	1	3	1	1	1	1	1	3	1	3	12	2	Tuberculoma	INFECTION
5	Thangapandi	35	M	2,5	2	1	1	2	3	3	1	1	1,2	3	2	3	4	1	1	1	1	1	1	2,3	1	3	1	CAD,HTN,CVA,Infarct	STROKE
6	Kavitha	20	F	3,5	2	2	2	3	7	2	1	1	0,4	1	1	1	2	1	1	1	1	1	1	1	1	12	2	Tuberculoma	INFECTION
7	Pandi	25	M	3	1	2	2	2	1	3	1	1	1	1	1	1	4	1	1	1	1	1	1	1	3	13	4	Primary brain tumour	TUMOUR
8	Michelraj	38	M	3	1	2	2	2	3	3	1	1	1,2,4	7	1	1	3	1	1	1	1	1	3	1	4	4	2	AIDS ,Tuberculoma	INFECTION
9	Rajendran	62	M	1	1	1	1	1	5	3	1	1	0	1	1	1	4	1	1	1	1	1	1	1	3	2	9,11	Cavernous heangioma,with heamorhage	STROKE
10	Laser	55	M	3	1	2	2	2	5	3	1	1	1,2	6	1	1	3	1	1	1	1	1	2	1	1	6	5	CA Lung ,sec brain	TUMOUR
11	Raman	60	M	3,5	1	1	1	2	1	3	1	1	1	1	1	1	4	1	1	1	2	1	1	1	2	1	1	Metabolic,hyponatremia,hypothyroidism	METABOLIC
12	Kannan	55	M	2	1	1	1	3	1	3	1	1	0	2,3	1	3	4	2	1	1	2	1	1	1	2	1	1	Hyperglycemic hyperosmolar state	METABOLIC
13	Krishnaveni	15	F	1	1	1	1	1	1	2	1	1	4	1	1	1	3	1	1	1	1	1	1	1	4	3	7	AVM with infarction	AVM
14	Vasuki	60	F	1	1	1	1	2	1	2	1	1	0	1	1	3	2	1	3	3	1	1	1	2	2	1	1	CKD Uremia	METABOLIC
15	Andi	60	M	3	1	1	1	3	1	3	1	1	1,2	1	1	1	3	2	1	1	1	1	1	1	1	1	1	Hyperglycemic hyperosmolar state	METABOLIC
16	Kannan	38	M	3	1	2	2	2	1	3	1	1	1,2,4	7	1	2	4	1	1	1	1	1	3	1	3	4	2	Tuberculoma,AIDS	INFECTION
17	Perasamy	62	M	3	1	1	1	2	3	3	1	1	3	1,2	1	1	3	1	1	1	2	1	1	1	1	1	1	Hyponatremia	METABOLIC
18	Mani	23	M	3	1	1	1	1	1	1	1	1	0	1	1	1	4	1	1	1	1	1	1	1	1	1	1	Cypermethrin poisoning	METABOLIC
19	Nagarathnam	32	M	3	1	1	1	2	3	3	1	1	1	3	1	3	4	1	1	1	1	1	1	1	1	3	1	CVA,old infarct ,hemiplegia	STROKE
20	Murugan	23	M	2	1	1	1	2	1	3	1	1	4	1	1	1	4	1	1	1	1	1	1	1	3	14	1	Single calcified lesion	CACIFIED GRANULOMA
21	Ananthan	26	M	3,5	1	1	1	2	1	3	1	1	1	0	1	1	4	1	1	1	1	1	1	1	3	14	1	Single calcified lesion	CACIFIED GRANULOMA
22	Veerammal	22	F	2	1	1	1	2	1	2	1	1	0	0	1	1	1		1	1	1	1	1	1	1	4	2	Tuberculoma	INFECTION
23	Ayavu	32	M	1	1	1	1	1	1	3	1	1	0	1	1	1	3	1	1	1	1	1	1	1	4	14	1	Single calcified lesion	CACIFIED GRANULOMA
24	Subunisha	23	F	3,5	2	2	2	3	3	2	1	1	4	1	3	1	3	1	1	1	1	1	1	1	4	14	8	Encephalitis ,old calcified granuloma	INFECTION
25	Muthuraman	35	M	3,5	1	2	1	2	1	3	1	2	1,2	2,5	3	1	4	2	1	1	1	1	1	1	3	7,8,11	6,10	CSOM, brain abcess,CVT	CVT,INFECTION
26	Chellammal	45	F	4	1	1	1	1	1	2	1	1	0	1	2	4	2	1	2	2	1	1	1	2	1	1	1	CKD Uremia	METABOLIC
27	Marriappan	63	M	3	1	1	2	3	2	3	1	1	1,2	2,3	2,3	3	4	1	1	1	1	1	1	2	1	2,3	11	CVA ,stroke	STROKE
28	Alagar	58	M	3,5	1	1	1	3	2	3	1	1	1,2	2,3	2	4	4	2	1	1	1	1	1	2	1	2	1	CVA ,stroke ,Heamorhage	STROKE
29	Yasothai	50	F	2	1	1	1	2	1	2	1	1	0	3	2	3	2	1	2	2	2	1	1	2	1	1	1	CKD Uremia,Hyponatremia	METABOLIC
30	Sankarapandi	15	M	3	1	1	1	2	1	3	1	1	0	1	1	1	4	1	1	1	1	1	1	1	3	5	3	Neurocysticercosis	INFECTION

31	Eswari	26	F	3,5	1	2	2	3	1	1	1	1	0	1	3	1	3	1	1	1	1	1	1	1	1	6	CVT,SSS thrombosis,postpartum	CVT
32	Danushkodi	34	M	3,5	1	2	2	3	1	3	1	2	3	1	3	1	3	1	1	1	1	1	1	1	4	7,8,11	6,10	CVT,brain abcess,CSOM	CVT INFECTION
33	Vellachamy	50	M	3,5	2	2	2	3	1	3	1	1	0	1	1	1	4	1	1	1	1	1	1	1	3	10	1	Osteomyelitis,epidural abcess,cerebritis	INFECTION
34	Pooja	21	F	3,5	1	2	2	2	1	1	1	1	0	1	3	1	3	1	1	1	1	1	1	1	4	3,8	1	CVT,SSSand cortical vein thrombosis postpartur	CVT
35	Raja	48	M	3	1	1	1	2	1	3	1	1	0	1	3	1	4	1	1	1	1	1	1	1	1	5	3	Neurocysticercosis	INFECTION
36	Swaminathan	42	M	2	1	1	1	2	1	3	2	1	2	1	1	1	4	1	1	1	1	1	1	1	1	9	1	Arachnoid cyst	Arachnoid cyst
37	Ramu	40	M	3,5	1	1	1	3	3	3	1	2	3	1	3	1	4	1	1	1	1	1	1	1	3	8,11	6	CVT, SSS,Transverse,sigmoid ,CSOM	CVT,INFECTION
38	Lakshmi	34	F	3	1	2	2	2	1	2	1	1	0	1	3	1	3	1	1	1	1	1	1	1	3	2,13,15	1	Brain tumour ,astrocytoma	TUMOUR
39	Ganesan	57	M	3	1	2	2	3	3	3	1	1	2	6	1	1	4	1	1	1	1	1	1	1	3	13,15	4	Brain tumour ,Glioblastoma multiforme	TUMOUR
40	sankaran	53	M	1	1	1	1	1	1	3	1	1	0	1	1	1	4	1	1	1	1	1	1	1	1	14	1	Single calcified lesion	CACIFIED GRANULOMA
41	Sudha	14	F	3	2	2	2	2	3	2	1	1	0	1	1	1	3	1	1	1	1	1	1	1	1	15	8	Encephalitis	INFECTION
42	Pandiammal	29	F	2	1	1	1	1	1	2	1	1	4	1	1	1	3	1	1	1	1	1	1	1	1	14	1	Single calcified lesion	CACIFIED GRANULOMA
43	maheswari	18	F	3	2	2	2	2	1	2	1	1	4	1	1	1	3	1	1	1	1	1	1	4	1	8	Encephalitis	INFECTION
44	Naallammal	72	F	3	1	1	1	2	1	2	1	1	1	2,3	2	4	2	2	2	2	2	1	1	2	1	1	1	Hyponatremia,CKD	METABOLIC
45	Priya	13	F	3	1	1	1	2	3	2	1	1	0	1	1	1	4	1	1	1	1	1	1	1	1	2	1	Heamorhagic stroke ,Thrombocytopenia	STROKE
46	Jaya	30	F	3	1	1	1	1	1	2	1	1	4	1	1	1	3	1	1	1	1	1	1	1	3	5	3	Neurocysticercosis	INFECTION
47	Panchavarnam	37	F	3	1	2	2	2	1	2	1	1	1	1	3	1	3	1	1	1	1	1	1	1	4	2,8,15	6	CVT,cortical vein thombosis	CVT
48	Senthil	22	M	3	1	2	2	2	4	3	1	1	4,1	1	3	1	4	1	1	1	1	1	1	1	4	8	6	CVT,cortical vein thombosis	CVT
49	Sankili	30	M	3	2	2	2	2	5	3	1	1	3	1	1	1	4	1	1	1	1	1	1	4	15	8	Encephalitis	INFECTION
50	Kamatchi	30	F	2	1	2	2	3	3	2	1	1	0	1	3	1	3	1	1	1	1	1	1	1	1	8,15	6	CVT,cortical vein thombosis	CVT
51	Thangamal	60	F	2	1	1	1	2	3	2	1	1	2	2,3	2	3	3	1	1	1	1	1	1	1	1	2	1	Heamorhage,CVA	STROKE
52	Meena	45	F	3	1	1	1	2	1	2	1	1	4	1	1	1	3	1	1	1	1	1	1	1	1	12	2	Tuberculoma	INFECTION
53	Balan	30	M	2	1	1	1	2	1	3	1	1	2,3	1	1	1	4	1	1	1	1	1	1		1	12	3	Neurocysticercosis	INFECTION
54	Babu	26	M	2	1	2	1	2	1	3	1	1	0	1	1	1	3	1	1	1	1	1	1	1	1	13	4	Brain tumour ,primary	TUMOUR
55	Bagavathi	35	F	3	1	1	1	1	1	2	1	1	4	1	1	1	3	1	1	1	1	1	1	1	1	14	1	Single calcified lesion	CACIFIED GRANULOMA
56	Durga	25	F	2	1	1	1	2	1	2	1	1	4	1	1	1	3	1	1	1	1	1	1	1	1	1	7	Cerebral AVM	AVM
57	Pandian	68	M	3,5	1	1	1	2	1	3	1	1	1,2	3	1	1	3	1	1	1	1	1	1	1	2	16	1	Alzheimers disease	DEGENERATION
58	Krishnan	52	M	3	1	1	1	1	1	3	1	1	1	2,3	2	2	4	3	1	1	1	1	1	1	2	1	1	Hypoglycemia	METABOLIC
59	Rajamani	45	F	4	1	1	1	2	1	2	1	1	0	1	1	3	2	1	3	3	1	1	1	1	1	1	1	CKD,Uremia	METABOLIC
60	chinnan	25	M	3	1	1	1	3	1	3	1	1	0	1	1	1	4	1	1	1	1	1	1	1	1	1	1	OPC poisoning,dimethoate	METABOLIC
61	kumar	25	M	3	1	1	1	2	1	3	1	1	1,2	1	1	1	4	1	1	1	1	1	1	1	1	1	1	Alcohol withdrawal	METABOLIC
62	Pothuponnu	69	F	3	1	1	1	2	6	2	1	1	1	6	1	2	3	1	1	1	1	1	1	1	4	6	5	Secondary brain ,ca breast	TUMOUR
63	Rathinam	72	F	3	1	1	1	2	3	2	1	1	1,4	1,2	2	4	3	1	1	1	1	1	1	2	1	2	1	Heamorhagic stroke	STROKE
64	Rani	38	F	2	1	1	1	2	3	2	1	1	0	8	1	1	3	1	1	1	1	1	4	4	1	3	1	CVA,cardioembolic ,	STROKE
65	Karthi	24	F	3	1	1	1	2	1	3	1	1	0	1	1	1	3	1	1	1	1	1	1	3	1	4	1	Tuberculoma	INFECTION
66	Muthu	44	M	2	1	1	1	1	1	3	1	1	4	1	1	1	3	1	1	1	1	1	1	3	12	3	Neurocysticercosis	INFECTION
67	marriammal	65	F	4	1	1	1	3	1	3	1	1	2,4	2,3	2	3	3	3	2	2	1	1	1	2	2	1	1	Hypoglycemia	METABOLIC
68	vasu	19	M	3	1	1	1	2	1	3	1	1	1	1	1	1	4	1	1	1	1	1	1	1	1	1	1	Cypermethrin poisoning	METABOLIC
69	Pechiammal	30	F	3	1	1	1	2	1	2	1	1	4	1	1	1	3	1	1	1	1	1	1	1	1	4	2	Tuberculoma	INFECTION
70	perumal	45	M	3	1	2	2	2	1	3	1	1	1,2	1	3	1	4	1	1	1	1	1	1	1	3	13	4	Primary brain tumour	TUMOUR
71	Lalitha	23	F	1	1	1	1	1	1	2	1	1	1	1	1	1	2	1	1	1	1	1	1	1	1	14	1	Single calcified lesion	CACIFIED GRANULOMA
72	sathya	34	M	1	1	1	1	1	1	3	1	1	0	1	2	2	2	1	2	2	1	1	1	2	1	1	1	CKD,Uremia	METABOLIC
73	Balan	32	M	3	1	1	1	2	1	3	1	1	1,2	1	1	1	4	1	1	1	1	1	1	1	1	1	1	Alcohol withdrawal	METABOLIC
74	Ahammed	38	M	3	2	2	2	3	1	3	1	1	1,2	1	3	1	4	1	1	1	1	1	1	1	4	7	1	Brain abcess	INFECTION
75	Sindhu	28	F	2	1	1	1	2	1	2	1	1	0	1	1	1	3	1	1	1	1	1	1	1	1	14	1	Single calcified lesion	CACIFIED GRANULOMA
76	Jaya	18	F	3	2	2	2	3	7	3	1	1	4	1	3	1	3	1	1	1	1	1	1	1	1	15	12	TB meningitis	INFECTION

77	Ravi	25	M	2	1	1	1	2	1	3	1	1	1	1	1	1	4	1	1	1	1	1	1	1	1	14	1	Single calcified lesion	CACIFIED GRANULOMA
78	Chandra	60	F	2	1	2	2	2	1	2	1	1	1	6	1	1	3	1	1	1	1	1	2	1	3	6	1	Secondary brain ,ca breast	TUMOUR
79	Sami	14	M	3	2	1	1	2	3	3	1	1	4	8	1	1	3	1	1	1	1	1	4	1	3	7	1	Brain abcess,CCHD,TOF	INFECTION
80	Saravanan	30	M	1	1	1	1	2	1	3	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	OPC poisoning,dimethoate	METABOLIC
81	Karuppaya	58	M	4	1	1	1	2	1	3	1	1	1,2	9	2	2	2	1	2	2	1	1	1	2	1	1	1	CKD,Uremia	METABOLIC
82	Ramuthai	68	F	3,5	1	1	2	3	1	2	1	1	0	2	2	2	3	2	1	1	1	1	1	3	1	1	1	Hyperglycemic hyperosmolar state	METABOLIC
83	Gopalan	65	m	1	1	1	2	2	1	3	1	1	1,2	9	2	2	2	1	3	3	1	1	3	3	2	1	1	CKD,Uremia	METABOLIC
84	Jayakumar	52	M	3	1	1	1	2	1	3	1	1	1,2	1	1	1	4	1	1	1	1	1	2	1	1	4	2	Tuberculoma	INFECTION
85	Ayyanar	23	M	3,5	1	2	2	2	3	3	1	1	1	1	3	1	4	1	1	1	1	1	1	1	1	8	6	CVT,cortical vein thombosis	CVT
86	Leela	15	F	3,5	1	2	2	3	3	2	1	1	0	1	3	1	3	1	1	1	1	1	1	1	1	8	6	CVT,cortical vein thombosis	CVT
87	Maniappan	29	M	2	1	2	1	2	2	2	1	1	1	1	3	1	4	1	1	1	1	1	1	1	3	7	10	Brain abcess	INFECTION
88	Thankamal	64	F	3	1	1	1	2	3	3	1	1	1	6	3	1	2	1	1	1	1	1	2	1	1	6	1	Secondary brain ,ca Lung	TUMOUR
89	Chinnakannu	56	F	3	1	1	1	2	1	2	1	1	1	1	1	1	3	1	1	1	1	1	1	1	3	4	2	Tuberculoma	INFECTION
90	marriammal	42	F	1	1	1	1	2	1	2	1	1	1	9	2	2	2	1	2	2	1	1	1	2	1	1	1	CKD,Uremia	METABOLIC
91	Ayyappan	72	M	3,5	1	1	1	3	1	3	1	1	0	2,3	2	1	4	2	1	1	2	1	4	2	2	1	1	Hyperglycemia,hyponatremia	METABOLIC
92	Kaliappan	42	M	2	1	1	1	2	1	3	1	1	1,2	1	1	1	4	1	1	1	1	1	1	1	3	12	2	Tuberculoma	INFECTION
93	Alagar	31	M	1	1	1	1	2	1	3	1	1	1,2	1	1	1	4	1	1	1	1	1	1	1	3	5	1	Neurocysticercosis	INFECTION
94	Kannamal	55	F	3	1	1	1	2	1	3	1	1	0	1	1	1	3	1	1	1	2	1	1	1	2	1	1	Hyponatremia,hypothyroidism	METABOLIC
95	Rajalakshmi	36	F	2	1	1	1	2	1	2	1	1	4	1	1	1	3	1	1	1	1	1	1	1	1	5	1	Neurocysticercosis	INFECTION
96	Das	55	m	3	1	1	2	2	1	3	1	1	1,2	4	1	1	3	1	1	1	1	1	3	1	1	12	2	Tuberculoma	INFECTION
97	Lakshmi	65	F	3	1	1	2	2	1	2	1	1	1	2,9	2	2	3	2	2	2	1	1	4	2	1	1	1	CKD,Uremia	METABOLIC
98	Priya	21	F	3,5	1	2	2	3	2	1	1	1	0	1	3	1	2	1	1	1	1	1	1	1	4	8,15	6	CVT,SSS,Cotical vein thrombosis ,postpartum	CVT
99	Perumal	45	M	3,5	2	2	2	3	2	3	1	1	1,2	1	3	1	3	1	1	1	1	1	1	1	3	7,15	1	Brain abcess	INFECTION
100	sundar	32	M	2	1	1	1	2	1	3	1	1	1,2	1	1	1	4	1	1	1	1	1	1	1	1	14	1	Single calcified lesion	CACIFIED GRANULOMA